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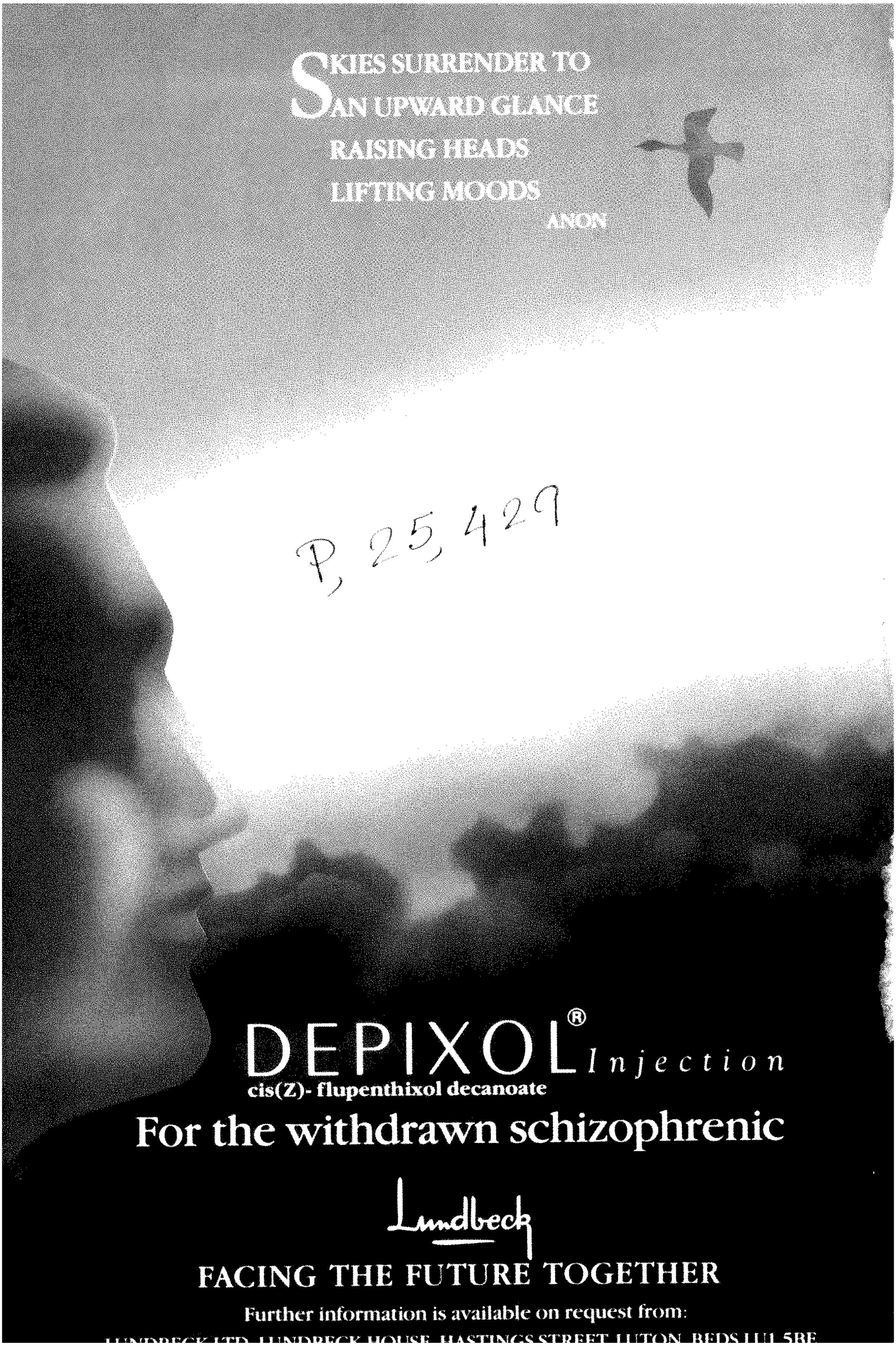

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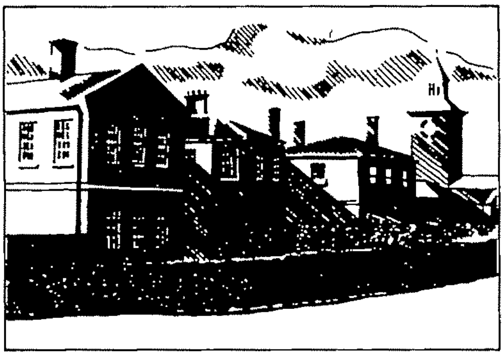

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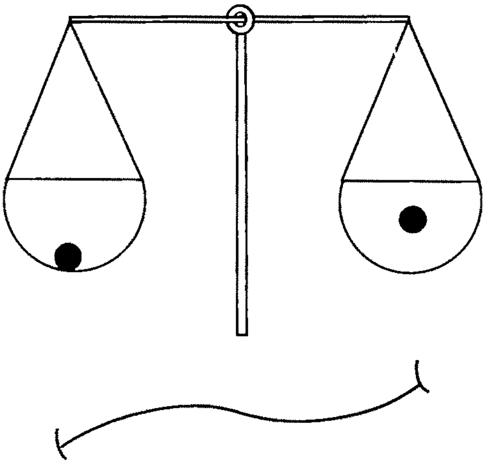

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Review Article

Genetic Linkage in Mental Illness

Limitations and Prospects

MIRON BARON, JEAN ENDICOTT and JURG OTT

Advances in genetic linkage strategies, including techniques of molecular genetics, augur well for the discovery of disease-related genes in mental disorders. Recent studies showing linkage of chromosomal loci to bipolar affective illness and schizophrenia attest to the potential in the 'new genetics'. However, the failure to replicate some of the early findings has led to calls for re-evaluation of the methodology in psychiatric research. Problems in studying complex (psychiatric) disorders include diagnostic uncertainties, unclear mode of transmission, aetiological heterogeneity, cohort effects, and assortative mating. Knowing the potential pitfalls in linkage analysis of mental illness should avert spurious findings and will increase the prospects of success.

The evidence for genetic factors in mental illness stems primarily from family, twin and adoption studies. Other methods of investigation include segregation analysis (a method of fitting probability models that test specific genetic hypotheses – e.g. single major gene or multifactorial/polygenic transmission – to family data) and studies with biological 'markers' (e.g. neurotransmitter enzymes and metabolites; receptor proteins; indices of brain imaging), and these have been used to search for mode of inheritance and biological susceptibility. Schizophrenia and affective disorders have been studied most vigorously using these approaches, but the results have not been consistent or readily interpretable (Baron, 1986*a*,*b*; Gershon *et al*, 1989).

Recent advances in molecular biology have rejuvenated psychiatric genetics and have renewed hope for the discovery of disease-related genes. At present the most commonly used approach in conjunction with molecular genetic techniques is linkage analysis. Linkage refers to the tendency of genetic variations (or alleles) at different chromosomal loci to be inherited together – two alleles that are close to each other on the chromosome are more likely to be inherited together than two that are further apart. Thus, genetic variations can serve as linked markers for other genes by 'tagging' their presence and approximate location. Particularly useful genetic variations in the laboratory are restriction-fragment-length polymorphisms (RFLPs) – restriction enzymes are used to cleave DNA at specific sites, and the length of the various fragments generated in this way (determined by the inheritance of the spacing of these sites on the DNA chain) are measured by gel electrophoresis. These 'new-generation' markers (RFLPs) span the entire human genome and supplant the 'classical' markers (e.g. leucocyte and red blood cell antigens; serum proteins) with the sheer force of numbers and degree of polymorphism (genetic variation). Highly polymorphic markers (i.e. loci with multiple alleles) are considered more useful for linkage analysis than markers with small genetic variation.

The genes for numerous hereditary conditions, including the neuropsychiatric disorders Huntington's disease and a familial form of Alzheimer's dementia, have already been localised using these techniques. Knowledge of the gene's location should enhance the precision of diagnosis, and allow screening. When the defective gene is isolated and its product identified, definitive prevention and treatment measures could be devised. The implications of these emerging technologies for psychiatry are discussed in detail elsewhere (Gurling, 1985, 1986; Baron & Rainer, 1988; Baron 1989*a*,*b*; Mullan & Murray, 1989).

The recent reports of linkage between bipolar affective illness and genetic markers on the X-chromosome (Baron *et al*, 1987; Mendlewicz *et al*, 1987) and chromosome 11 (Egeland *et al*, 1987), and between schizophrenia and genetic markers on chromosome 5 (Sherrington *et al*, 1988), attest to the potential of the genetic marker strategy. However, reports of pedigrees unlinked to either set of markers have somewhat tempered the early enthusiasm. Specifically, attempts at replicating the chromosome-11 and chromosome-5 linkage results have not been successful (Hodkinson *et al*, 1987; Detera-Wadleigh *et al*, 1987; Gerhard *et al*, 1988; Gill *et al*, 1988; St Clair *et al*, 1989; Kennedy *et al*, 1988; Diehl *et al*, 1989; Kaufmann *et al*, 1989; Aschauer-Treiber *et al*, 1990). Furthermore, on extension and reanalysis of the pedigree data, the evidence of linkage between bipolar illness and chromosome-11 markers was substantially reduced (Kelsoe *et al*, 1989). X-linkage, on the other hand, has been reported by several investigators, although unlinked pedigrees are clearly present (see reviews by Risch & Baron, 1982; Baron, 1989*a*; Berrettini *et al*, 1990). Ongoing studies aim to extend and refine the X-linkage findings (Baron, 1989*a*; Freimer *et al*, 1989).

The flurry of claims and counterclaims has engendered calls for re-evaluation of genetic linkage strategies in psychiatry. Although the diverse results can be ascribed to aetiological heterogeneity, the possibility of false-positive and false-negative linkage results must be considered. The resolution of this issue is critical to the viability of the newly fostered link between the 'new genetics' and psychiatry.

In this article we discuss methodological and analytical questions central to the design and interpretation of linkage studies in mental illness. We then propose guidelines for standardised procedures in this area of research. We contend that a sober view of potential pitfalls in linkage analysis of complex disorders is crucial to the resolution of current controversies. Some of these issues were reviewed elsewhere (Baron, 1986*b*, 1989*a*; Blehar *et al*, 1988; McGuffin, 1988; Rice & Risch, 1989), but before the recent linkage studies and not to the extent of this review.

Linkage analysis

The major approaches to linkage analysis were reviewed by Ott (1985). There are several issues that need to be addressed in performing linkage analysis of complex disorders such as mental illness. These include mode of inheritance, phenotypic uncertainties, cohort effect, aetiological heterogeneity, and the confounding effect of multiple tests.

Mode of inheritance

The assumption underlying linkage analysis is that the illness is caused by a single diallelic Mendelian locus. The disease locus may display incomplete penetrance (the probability of manifesting the phenotype given the genotype), which is age dependent. Using the lod-score method (a statistical procedure based on the odds ratio, i.e. the ratio between the probability of observing the data under the hypothesis of linkage and that of there being no linkage; the ratio is commonly expressed as its logarithm, or lod score), one must specify mode of inheritance parameters, such as gene frequency and penetrances. For age-dependent penetrance, one must also specify an age-at-onset function.

These assumptions may be violated in a number of ways. Firstly, in the light of aetiological heterogeneity (i.e. different genetic and non-genetic mechanisms underlying different subsets of the illness) and other complicating issues, we will not know with precision what are the correct mode-of-inheritance parameters for a given pedigree. The usual practice is to use a few different sets of parameters to determine whether linkage exists. The effect of misspecifying mode-of-inheritance parameters is well known. The primary effect is to reduce the expected lod score and increase the estimated value of the recombination fraction. (Recombination refers to a rearrangement of alleles in offspring, due to exchange of material between homologous chromosomes during meiosis; the closer the loci are to each other, the less likely is recombination between them, and thus the distance between loci can be inferred from the recombination fraction.) Therefore, it is not expected that parameter misspecification will lead to false evidence for linkage (Ott, 1985; Clerget-Darpoux *et al*, 1986).

Secondly, the true mode of inheritance in a given family may involve more than one locus – for example, a disease may result from the interaction of alleles at more than one locus (i.e. joint action of a small number of gene loci, or oligogenic mode of inheritance). However, even if this was true, it may still be feasible to detect the contributing loci through linkage analysis assuming a single Mendelian locus (Majumder, 1989). This has been demonstrated through linkage studies of diseases associated with histocompatibility locus antigens (HLA) which have detected disease-susceptibility loci with only minor effects on the familial aggregation of the disease (Suarez *et al*, 1982; Risch, 1987). Again, the primary effect under these circumstances would be a reduction in the expected lod score and an overestimation of the recombination fraction. For example, one might assume that three gene loci interact to lead to schizophrenia, all three being unlinked to each other, and only one of them being linked to a given marker. Depending on the assumed model of interaction among the trait-causing loci, existing computer programs can be modified to allow for this type of linkage analysis.

An alternative to lod-score analysis is the affected-sib-pair method (Suarez *et al*, 1982; Lange, 1986). According to this method, pairs of affected siblings will be more likely to have the same marker than expected by chance if linkage exists between the illness and the marker locus. Unlike the lod-score approach, sib-pair analysis requires no assumptions about mode of inheritance and genetic parameters (e.g. penetrance). Therefore, it may be less prone to spurious linkage results, especially false-negative findings. However, the method is considered less efficient and less powerful than lod-score analysis, especially in the presence of significant genetic heterogeneity. Recently, the sib-pair method was modified to be used in conjunction with lod-score analysis (Sandkuyl, personal communication). This 'extended' sib-pair method is thought to be more versatile in handling genetic information. Sib-pair analysis appears particularly useful when the affected subjects are concentrated in one generation only, a scenario likely to occur in some mental disorders, such as schizophrenia.

Phenotypic uncertainties

An accurate definition of the phenotype is crucial in linkage studies. This issue is accorded special importance in psychiatric diagnosis, where the lack of external validating criteria, the considerable variation in form and severity of the clinical manifestations, and the likely presence of phenocopies (cases unrelated to the genotype tested) complicate diagnosis.

Phenotypic misclassifications can lead to spurious linkage findings and to biased estimates of genetic parameters (Ott, 1977; Suarez *et al*, 1987; Martinez *et al*, 1989). Barring a systematic error in the assignment of phenotypes, this type of error would not, by and large, generate false evidence of linkage, especially if the misclassification entails false positives (cases erroneously classified as affected). However, it may lead to false-negative results by missing genuine linkage. False negatives (cases misclassified as unaffected) can generally be managed in the context of incomplete penetrance. However, if the estimated penetrance value is high, false negatives can have an impact on the linkage results, especially if the misclassified individuals are well into the risk period. Thus, the critical role of adequate diagnostic procedures cannot be overemphasised.

Defining phenotypic boundaries for linkage studies is a complex task. The classic criterion is the extent to which potentially related clinical states show familial aggregation. This criterion has guided most of the recent linkage studies. However, while the 'core' phenotype (e.g. bipolar I disorder in affective illness; chronic schizophrenia in schizophrenic disorders) may be considered 'true' representations of the genotype by virtue of their severity, diagnostic reliability and presumed homogeneity, the milder, more common conditions (e.g. bipolar II disorder, unipolar depression, cyclothymia, hypomania and minor depression in affective disorders; acute

TABLE I

Phenotypic classifications for pedigrees with bipolar affective disorder

Phenotype	*Diagnostic boundaries*		
	Narrow	*Intermediate*	*Broad*
Affected	BPI; manic; SA-manic	Add to 'Narrow' BPII (with major depression); recurrent UP depression; recurrent SA-depressed	Add to 'Intermediate': BPII (with minor or intermittent depression); single episode UP depression; single episode SA-depressed; cyclothymia; hypomania
Uncertain	BPII (all types); UP depression (all types); SA-depressed; schizophrenia; unspecified psychosis; minor affective disorders	BPII (with minor or intermittent depression); single episode UP depression; single episode SA-depressed; schizophrenia; unspecified psychosis; minor affective disorders	Schizophrenia; unspecified psychosis; minor affective disorders (other than hypomania and cyclothymia)
Unaffected	All other diagnoses, including normal	All other diagnoses, including normal	All other diagnoses, including normal

BP: bipolar; SA: schizoaffective; UP: unipolar. Minor affective disorders: hypomania, cyclothymia, labile personality, minor depression–recurrent and chronic, intermittent depression. 'Uncertain' cases are to be treated as unknown in the linkage analysis. Diagnostic classifications are based on the Research Diagnostic Criteria . For clarity and brevity, only diagnoses at the definite or probable level were considered. The proposed phenotypes are based on current notions concerning familial aggregation; however, opinions may vary as to the 'precise' diagnostic boundaries in the various categories.

schizophrenic-like psychosis, schizotypal, paranoid and schizoid personality disorders in schizophrenia) are viewed as a heterogeneous group of disorders which are likely to include a sizeable number of diagnostic misclassifications (false positives, or phenocopies).

To redress this potential impediment, three strategies can be employed. Firstly, as proposed in Table I, the 'affected' category in families of probands can be varied from conservative, or narrow definitions, to much broader sets of criteria. This approach, which takes into account the severity, reliability and stability of the diagnosis, examines to what extent the different clinical manifestations are an expression of the putative disease genotype. By focusing initially on the 'core' phenotype, which is less susceptible to diagnostic uncertainties, linkage results can be lent more credence. For example, the X-linkage finding in bipolar illness (Baron *et al*, 1987) is robust over a range of phenotypic patterns, including a narrow definition of the phenotype (Baron *et al*, 1990). However, the evidence of linkage between schizophrenia and chromosome-5 markers is diminished considerably using the narrow definition of the illness (Sherrington *et al*, 1988).

Secondly, estimates of diagnostic uncertainty and phenocopy probability can be incorporated in the analysis to enhance the robustness of the linkage results. Such estimates can derive, for example, from clinical characteristics that predict or reflect the stability and severity of the psychiatric condition (e.g. recurrence of episodes, number of symptoms, history of treatment; Rice *et al*, 1987) or from the quantity and quality of the clinical data upon which the diagnosis is based. For example, a diagnosis based on concordance between all sources of information (e.g. personal interview, family history, and medical records) may be given a higher 'score' of certainty than a diagnosis based on more limited information (e.g. family history). This can be accomplished by introducing separate penetrance classes that reflect the probability of having observed the phenotype given each of the possible genotypes. This approach is described in detail elsewhere (Baron *et al*, 1990).

Thirdly, a 'penetrance-free' model can be used, in which unaffected individuals are excluded from the analysis. This stringent approach obviates the question of false negatives and is robust with respect to incomplete penetrance. When applied to the linkage data on schizophrenia and chromosome-5 markers, the lod score fell short of statistical significance (Sherrington *et al*, 1988).

Once linkage is found, other diagnostic boundaries can be employed to determine how they influence the linkage statistic. However, to avert spurious linkage results, decisions concerning phenotypic definitions must be made before the linkage analysis. In our opinion, these definitions should not deviate significantly (at least in the first round of analyses) from generally accepted notions of familial aggregation. For example, a cluster combining disorders thought to be distinct (e.g. affective illness and schizophrenia) subsumed under the same phenotypic rubric will likely undermine the accuracy of linkage analysis. The extent to which unconventional phenotypic definitions affected some of the recent linkage results is unclear (Sherrington *et al*, 1988; St Clair *et al*, 1989).

Cohort effect

A third problem relates to the possibility of a secular trend (cohort effect). A change in the rate of mental illness over time, or across generations, has been best documented for affective disorder (Klerman *et al*, 1985; Risch *et al*, 1986). This effect has been attributed to selective recall among older individuals, low accuracy of data obtained on deceased relatives, increased environmental effects, or unknown causes.

This issue is akin to variable age at onset with regard to linkage analysis. According to a single-locus, two allele model, there are three possible genotypes, and a penetrance associated with each genotype. In a standard linkage analysis, these penetrances can be made to depend on age. In a similar way, the three penetrances can be made to depend on cohort (or year of birth), depending on the perceived cause of the cohort effect. For example, with regard to a single-locus model, there are two most likely explanations: (a) the penetrances of the high-susceptibility genotype(s) is (are) changing over times, or (b) the penetrances of the low-susceptibility genotype(s) are changing over time (i.e. change in frequency of 'sporadic cases'). If the former explanation is correct, then we should observe no change in the 'heritability' over time, since the genotype distribution among the affected is not changing over time. Alternatively, if the second explanation is correct, then we would see a change in heritability over time. For example, if 'sporadic' cases were increasing in frequency with birth cohort, then we would expect that heritability would decrease with birth cohort. An appropriate adjustment can then be made in the linkage analysis by defining age-specific and cohort-specific penetrances. Because such an adjustment may not be precisely correct, it is important to consider what the impact may be of an incorrect cohort adjustment or no cohort adjustment at all.

By not including a cohort adjustment, the penetrances assumed for specific individuals may not be correct. Therefore, the overall impact should be similar to that described above for misspecifying mode-of-inheritance parameters – namely, a decrease in the expected lod score, and an increase in the estimated recombination fraction. A recent reanalysis of X-linkage data in bipolar affective illness indeed gave rise to an increased lod score when cohort effect was taken into account (Van Eerdewegh, 1989).

Aetiological heterogeneity

It is generally held that mental disorders are probably heterogeneous. Linkage analysis is recognised as a potentially powerful tool in unravelling genetic heterogeneity. Its usefulness has been demonstrated in several disorders with complex or practically unknown mode of transmission. Several tests of genetic linkage heterogeneity have been proposed, including likelihood ratio tests, Bayesian and non-Bayesian methods (Ott, 1985).

Performing linkage analysis in the presence of genetic heterogeneity should not lead to false evidence of linkage (Ott, 1985). On the contrary, under some circumstances, especially when the proportion of linked pedigrees is small, linkage may be missed entirely. Simulation studies have shown correspondence between the degree of heterogeneity and the number of families necessary to establish linkage for a complex disorder (Cavalli-Sforza & King, 1986; Ott, 1986*b*; Martinez & Goldin, 1989). For example, hundreds of nuclear families each with several affected members may be required to demonstrate linkage if only 10% of the cases are linked to a particular locus. Complete RFLP maps coupled with the availability of dense pedigrees could help reduce the sample needed to detect linkage.

A complete linkage map of the human genome is rapidly becoming a reality (Donis-Keller *et al*, 1989). Statistical methods, such as interval mapping and simultaneous search, that are designed to exploit the power of a genomic linkage map, have been developed (Lander & Botstein, 1986). Using interval mapping, one examines the hypothesis that a trait caused by a single genetic locus maps in the middle of the interval between an adjacent pair of RFLP cleavage sites. The linkage hypothesis is easier to test using this paradigm against an alternative scenario whereby the locus is linked to a single RFLP marker at an unknown distance. The method of simultaneous search involves studying the segregation of several candidate marker loci simultaneously to determine whether they can jointly explain the transmission of the disorder in the different families being studied. Further reduction in the number of families required to map heterogeneous disorders could be achieved by focusing on very large pedigrees, each sufficiently informative to show linkage independently of additional information. However, such pedigrees are rare.

A related question is whether one genetic type of the illness can be distinguished on clinical (e.g. phenomenology, course, treatment response) and biological (e.g. biochemical, neurophysiological, neuroanatomical) grounds. By comparing one genetic form of the disorder with other genetic or non-genetic forms, aetiological heterogeneity can be elucidated. This, in turn, might facilitate the search for other linked pedigrees by focusing on specific clinical and biological characteristics. A case in point is X-linked bipolar affective illness, which appears to display early age at onset (Baron *et al*, 1990). Examples from other branches of medicine include diabetes mellitus where clinical (early onset) and biological (insulin dependence) factors combine to identify a form of the illness that is associated with certain HLA haplotypes (the histocompatibility region of chromosome 6).

Multiple tests

An inherent danger in analysing disorders with unclear mode of inheritance and ambiguous phenotype is the temptation to maximise the lod score by varying both the values of the genetic parameters and the phenotypic classifications of disorder. Simulation studies have shown that this practice may lead to inflated lod scores and thus may carry the risk of producing false-positive results (Weeks *et al*, 1990). To avert spurious linkage results, decisions concerning the models tested should be made before the linkage analysis. Additionally, the range of parameter values and phenotypic definitions should be limited. Alternatively, methods accounting for inflated lod scores should be developed. It may also be advisable to make allowance for the effect of studying multiple linkage markers as another safeguard against chance findings (Ott, 1986*a*; Edwards, 1989).

The linkage statistic

Higher lod scores than the conventional value of 3.0 (an odds ratio of 1000:1 in favour of linkage over non-linkage) may be required to increase confidence in reported linkages. Since *a priori* the odds against any two loci being linked are 50:1, a lod score of 3.0 corresponds to *a posteriori* odds ratio of only 20:1 (i.e. an approximate 5% confidence level). While this level may be adequate for simple Mendelian traits, it may be found wanting for complex disorders.

Segregation analysis

Whether to perform segregation analysis before linkage analysis is a contested issue. In the context of linkage studies, the primary goal of segregation analysis is the identification of major locus effects and the estimation of genetic parameters. However, segregation analysis may be compromised by the same uncertainties that confound linkage analysis, most notably aetiological heterogeneity. The likely heterogeneous nature of mental disorders may explain the ambiguous results obtained of previous segregation analyses of these disorders (Baron, 1986*a*; Gershon *et al*, 1989). Additionally, segregation analysis requires systematic collection of family data. This is a costly proposition, especially when most of these families may not be studied for linkage (for instance, small families with low illness density). It is doubtful whether the added precision in the estimation of genetic parameters is worth the cost (given the uncertainties mentioned above, it is not at all clear whether an added precision can be attained).

In light of these considerations, some investigators cast doubt on the value of segregation analysis before the availability of information on linkage markers. Instead, should linkage be found, the parameter set might be varied within certain limits to study the robustness of the results. It is also possible to combine segregation and linkage analysis once linkage is detected.

Diagnostic procedures

The utility of psychiatric diagnosis has been enhanced considerably by operationalised diagnostic constructs, such as the Schedule for Affective Disorder and Schizophrenia (SADS; Endicott & Spitzer, 1978) and the Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978). The object of these diagnostic tools is to improve diagnostic reliability and to provide clinicians and investigators with a common language. Operational diagnostic instruments were used in all of the recent linkage studies. However, recent data indicate that while diagnostic reliability can be raised to an acceptable level within a given diagnostic centre, markedly disparate diagnostic practices may exist among different research teams using the same criteria (Zimmerman, 1988). This does not augur well for the reliability of linkage data. Appropriate training and research designs need to be implemented by experienced investigators, with due attention to repeated observations and quality control. High levels of diagnostic reliability are called for both within and between research groups. The latter can be accomplished by designating diagnostic centres with an established track record in the development and application of diagnostic procedures as sites for centralised training. Alternatively, the diagnostic material obtained in the course of genetic investigations could be subjected to independent assessment by experienced diagnosticians from other centres.

Clinical information

The clinical assessment should be based primarily on personal interviews supplemented by family history from multiple informants and medical records. 'Best-estimate' diagnoses can then be made by independent diagnosticians using all available sources of information. The ratings can be used to monitor and evaluate inter-rater reliability and to arrive at final consensus diagnoses. The use of multiple sources of information would minimise 'missed' diagnoses and enhance the accuracy of the clinical determination. It may also be advisable to gather clinical data applicable to several diagnostic systems. This should allow more latitude in assigning phenotypic boundaries and would facilitate a comparative analysis should the diagnostic systems vary among studies.

Blindness

Even if the various diagnostic procedures are impeccable, there are other sources of error that will likely lead to phenotypic misclassifications. One stems from carrying out the clinical assessment with prior knowledge of familial relationships and diagnostic status of other family members, which could lead to a bias toward overdiagnosis (false positives). To implement the ideal design, where complete blindness is observed, would require considerable resources (specifically, a large number of clinical interviewers). One way of circumventing this problem is to have the 'best-estimate' diagnoses done blind to family data. This, however, is only a partial solution, since the final diagnosis is contingent on the original clinical data and is therefore sensitive to the manner in which these data are collected. In addition, it is imperative to maintain the diagnostic procedures and the genetic marker determination independent of each other.

Linkage markers

Several categories of linkage markers can be considered:

(a) RFLPs related to candidate genes (genes with potentially relevant neurobiological function).

These include, for example, genes coding for neuroreceptor proteins, neurotransmitter enzymes, and receptors for psychoactive drugs; cDNA clones (clones made of complementary DNA that is synthesised from a messenger RNA template) that distinguish diseased from normal brain tissue and thereby appear to be disease-related; probes for gene expression in brain tissue using cloned protein products of specific brain mRNA.

(b) RFLPs localised in 'candidate' genomic regions.

For example, the co-occurrence of the illness and cytogenetic abnormalities (e.g. deletions, translocations) may point to the particular chromosomal aberration as a source for site-specific RFLPs. Using this approach, 'candidate' chromosomal sites on chromosome 5 and 11 have been proposed for schizophrenia (Bassett *et al*, 1988) and affective disorder (Smith *et al*, 1989). Another instance of 'candidate' chromosomal region derives from sex-dependent transmission patterns which could point to the sex chromosomes as potential locations for the disease gene. For example X-linkage in bipolar affective illness (see reviews by Baron *et al*, 1981; Risch & Baron, 1982) and pseudoautosomal susceptibility locus for schizophrenia (Collinge *et al*, 1989).

(c) Random, or abitrary RFLPs spread throughout the genome.

These different approaches can be used concomitantly to maximise the prospects of identifying disease-related genes. The rapid advance in constructing genetic linkage maps that span the human genome makes it likely that many genes of interest will be localised, identified, isolated, and cloned (Donis-Keller *et al*, 1989). Molecular genetic techniques that can be used to attain this goal are described in detail elsewhere (Davies, 1986; Gurling, 1985, 1986; Baron, 1989*a*,*b*; Baron & Rainer, 1988).

Whatever approach is taken, the typing of linkage markers must be made blind to the clinical information. In addition, RFLP patterns should be read and interpreted independently by different observers, and a consensus should be reached before the linkage analysis. If possible, RFLPs should be retyped in a different laboratory without prior knowledge of the original results. These measures should prove helpful both in establishing within- and between-laboratory reliability and in minimising other sources of error in gene marker determination.

Selection of pedigrees

Size and density

Extended multigenerational pedigrees are considered more useful for genetic marker studies than nuclear families, and larger pedigrees contain more information than smaller pedigrees. Sequential sampling schemes are considered most practical for studying a potentially genetic condition (Cannings & Thompson, 1977; Hodge & Boehnke, 1986). Generally, linkage results obtained with large pedigrees are less susceptible to ascertainment bias than small families.

To order family pedigrees according to their suitability for linkage analysis, the following procedures can be employed. Before typing, judgement will have to be based on the phenotypes at the disease locus. In a Mendelian trait with reduced penetrance, assuming absence of phenocopies, the underlying genotype is exhibited only in affected individuals. The total number of affected individuals in a pedigree may thus be taken as a measure for a pedigree's suitability for linkage analysis. It is, therefore, generally possible to judge by inspection which pedigrees are most suitable for further investigation. A more sophisticated treatment of this question has been offered by Boehnke & Ploughman (1986), who estimated by computer simulation the expected lod score in a pedigree, given the phenotypes at the disease locus.

For some mental disorders, such as bipolar affective illness and panic disorder, the task of obtaining large pedigrees with a high density of illness is not unduly difficult. In other mental conditions, especially schizophrenia, such pedigrees are hard to come by owing to reduced fertility and high geographic mobility. In these cases, alternative strategies may be required, such as a search for population isolates where large, dense pedigrees may exist, or the choice of smaller families in which sib-pair analysis can be employed (see under 'Linkage analysis'). Although small families are clearly inferior to large pedigrees in the presence of significant aetiological heterogeneity, they may, by virtue of their size, incur fewer opportunities for other complicating factors, such as phenocopies and assortative mating (see below).

The selection of family members for typing will be based on the potential to exhibit the genotype: the affected are more useful than the unaffected, and older unaffected persons are more useful than young unaffected members. Clearly, when a parental mating is not informative (e.g. when the parent through whom the illness is presumed to be transmitted is not doubly heterozygous, i.e. heterozygous at both the putative disease locus and the marker locus, a scenario necessary to elucidate the transmission pattern of the disease and marker alleles), then their children would contribute very little to the linkage results.

Assortative mating

Assortative mating may confound linkage analysis by increasing the extent of genetic heterogeneity within pedigrees, as the person marrying into the family may introduce a genetic type of the disorder different from the one already segregating in the pedigree. Assortative mating has been observed in mental illness, especially in affective disorder (Merikangas & Spiker, 1982; Merikangas *et al*, 1988) and, possibly, in schizophrenia (Baron *et al*, 1983). Also, an unaffected spouse may nevertheless be a carrier of the genetic disposition.

The overall effect of assortative mating would be to increase the rate of phenocopies for a particular gene and thereby diminish the evidence of linkage (i.e. a type II error) (see under 'Linkage analysis'). It is, therefore, recommended that spouses and their relatives be evaluated systematically, and that pedigrees, or branches thereof, with evidence of illness on both paternal and maternal sides (i.e. bilateral, or bilineal transmission) be excluded from the linkage analysis. How rigorous the exclusion criteria must be remains an open question. At the very minimum, the presence of the 'core phenotype' (e.g. bipolar I disorder or recurrent unipolar illness in affective disorder), either in the spouse or in the spouse's immediate family, should be sufficient for the exclusion of that portion of the pedigree.

X-linkage v. autosomal transmission

When X-linkage is examined, it is common practice to exclude pedigrees with male-male transmission. However, the remaining pedigrees may have to be weighted differently as a function of the number of opportunities to display male-male inheritance. Namely, pedigrees with few such opportunities (for instance, where informative males have no male offspring) and no evidence of X-linkage should be assigned a lower 'score' than those with many such opportunities. Thus, evidence against X-linkage in a sample comprised primarily of the first type of pedigrees would be less compelling than that obtained with the second category of pedigrees.

Replication

By and large, it is apparent that true linkage can be more easily missed than detected. Namely, incomplete penetrance, phenotypic misclassifications, cohort effect, and assortative mating would not inflate the lod score; rather, they might lead to false-negative linkage results. These factors must therefore be taken into account in the linkage analysis. To be sure, the types of error leading to negative linkage data are compounded by the low prior probability of detecting linkage in the presence of significant aetiological heterogeneity.

Ultimately, replication of linkage findings would be the desirable outcome. However, in the light of the methodological issues, this may take time. Meanwhile, pedigrees with reported linkages can be put to the test in several ways: (a) the study of multiple genetic markers in the candidate chromosomal region using the ever-improving genomic map; (b) systematic updating of diagnoses aimed at addressing the question of diagnostic stability, especially in the light of variable age at onset; and (c) extending the pedigrees with the aim of augmenting the genetic information. If the evidence for linkage holds up in the face of these measures, the validity of the original findings can be stated with greater confidence, and the prospects of eventual replication would seem brighter.

Summary and recommendations

If major genes operate in some mental disorders, they will likely be detected by genetic linkage strategies. Suggested guidelines that reflect the major methodological issues discussed in this article are summarised below.

Linkage analysis

(a) Estimates of mode-of inheritance parameters (i.e. allele frequencies, penetrance) should be 'realistic' and confined to a narrow range. Other pivotal factors, such as age at onset and cohort effect, should be incorporated in the analysis.

(b) The choice between the lod-score approach and sib-pair analysis should be based on the nature of the sample, with due attention to the advantages and drawbacks of each of these methods.

(c) Phenotypic uncertainties should be factored in the analysis by way of using: (i) a stepwise hierarchical approach, varying the phenotype from narrow to broad boundaries; (ii) estimates of diagnostic uncertainty and phenocopies based on the stability and severity of diagnosis and the informativeness of the diagnostic data; (iii) a 'penetrance-free' model of analysis. At our present state of knowledge, conservative definitions of the phenotype should be lent more credence than less well established diagnostic boundaries.

(d) Statistical methods that address the question of aetiological heterogeneity, with a view to sampling schemes and genomic linkage maps, should be considered in study design and data analysis.

(e) Clinical and biological indices that may identify homogeneous illness subsets should be sought and utilised in the sampling scheme.

(f) Decisions concerning models and phenotype definitions should be made before the linkage analysis. Generally, the number of tests and classifications should be limited. Allowance should be made for the effects of multiple testing, such as number of linkage markers.

(g) Stringent requirements concerning the linkage statistic are advised. Specifically, the critical lod score may have to be higher than the conventional value of 3.0.

Diagnostic procedures

(a) Standardised research diagnostic techniques with due attention to intra- and inter-centre reliability should be employed.

(b) The clinical information should be comprehensive and based on multiple sources.

(c) Diagnostic determinations should be made blind to genetic marker data. If possible, blindness should also be maintained as regards familial relationships and clinical status of other family members.

Linkage markers

(a) Various categories of linkage markers (candidate genes, candidate chromosomal regions, and random RFLPs) can be studied concomitantly.

(b) The typing of markers must be made blind to the clinical data.

(c) Reliability of marker typing should be established both within and between laboratories.

Selection of pedigrees

(a) The sampling scheme, or method of ascertainment, should be described.

(b) Generally, a premium should be placed on large, dense pedigrees. For some disorders, in which such pedigrees may be rare, smaller families and sib-pairs may be studied.

(c) Spouses marrying into the family and their relatives should be evaluated systematically. Pedigrees, or branches thereof, with evidence of assortative mating or bilineal transmission should be excluded.

Replication

(a) Independent replication is required as a 'proof' of linkage.

(b) Pedigrees with evidence of linkage should be further scrutinised by extending the diagnostic and genotypic information.

The phenotypic and genetic complexities of mental disorders will likely render the search for reproducible linkages an arduous undertaking. Standardised procedures that address methodological issues in the design and interpretation of linkage studies should expedite this process.

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Point of View

Projective Identification in General Psychiatry

JON JUREIDINI

Projective identification is a psychoanalytic concept encompassing functions of defence and communication. It is invaluable in a variety of settings in general psychiatry, including patient assessment, ward management and work with families.

As a trainee psychiatrist I was confused by the language that people used in speaking or writing about projective identification and by the seeming contradictions in various explanations. When trying to make sense of the concept, the literature was, with a few exceptions (e.g. Ogden, 1979; Moustaki, 1981; Casement, 1986), off-putting to someone not at ease with psychoanalytic jargon. However, I now believe that the mystery and complexity of projective identification are often exaggerated, in a way which unnecessarily inhibits broader exposure and discussion. Despite variations in the usage of the term, the concept is relevant and accessible to the general psychiatrist.

Demystifying projective identification

The various definitions of projective identification need not be mutually exclusive, but can instead describe different aspects of the same complex process. An analogy is when someone is described as 'depressed'. Grouping together recognisable cases of depression is logical and useful, but within the group there will be variations in aetiology, phenomenology, outcome and degree of overlap with other syndromes. Further information and formulation are therefore required to define any single episode of depression adequately. Similarly, on examining many examples of projective identification (e.g. Rosenfeld, 1965; Bion, 1967; Zinner & Shapiro, 1972; Segal, 1973), I found wide variations even between different examples quoted within a single article. There are: projection onto or projection into; variable degrees of splitting and denial in the process; projective identification as communication or as defence; and a range of responses elicited in the recipient.

Some authors (e.g. Meissner, 1987) have argued that the use of 'projective identification' should be restricted to the way Klein (1946) intended, lest it become an overinclusive term for all manner of interpersonal interactions. This approach is not helpful, however. There is a common theme which justifies the use of the term to describe these apparently disparate psychic functions. I begin by focusing on the variation in usage of the term and then attempt to draw together this unifying theme.

It is useful to think of projective identification as a complex process made up of a variable combination of simpler components. In fact, there is a case for labelling it 'projection and identification' rather than 'projective identification'. The projection is usually of some quality/emotion (e.g. fragility) which has first been split off from its complementary partner (e.g. strength) and denied in the self.

> For example: A husband may need to reinforce his precarious sense of his own strength by splitting off and denying any fragile qualities within himself and projecting them into his wife. He can then safely recognise these qualities and attend to them, because they now reinforce his strength rather than threatening him. If the wife behaves in accordance with the projections, the result may be adherence to rigid sex-role stereotypes within the marriage.

It is not, however, inevitable that projected qualities be totally split off and denied.

> For example: A student was enraged by a lecturer. He unconsciously attributed wounded feelings to the fact that a female student had, in his opinion, been wrongfully attacked by the lecturer. While he did not lose the capacity to acknowledge his own hurt and rage, he identified the wrong perceived to be done to the woman as the stimulus for his aggrieved protest.

The projection need not be into a single individual and can involve three or more people in a complex pattern of mutual projection and identification.

> For example: The complementary sex-role stereotyped couple described above had a teenage daughter who was perceived by the father as being fragile like her mother but by the mother as a potentially violent 'she-devil'. All three of them therefore acted as if the mother was indeed fragile and the father strong, thus reinforcing the stereotypes and supporting the vulnerable marriage. This left the daughter with the impossible task of identifying with both sets of projections.

In families where more than one child is available, complementary qualities can be projected into different children.

There are at least three qualitatively different ways in which the object of the projection can be involved in the process. At one extreme are cases in which the process takes place entirely independent of the target.

For example: A worker believed that he had been wrongfully denied promotion because of a company director's racial prejudice. Yet objectively the worker had been fairly treated and the director was neither prejudiced nor involved in the decision about promotion.

In such cases, neither party identifies with aspects of the other. Projection might be a more appropriate term than projective identification here, although there is vigorous disagreement in the literature about the relationship between the two concepts.

The intermediate way in which the object of projection is involved seems closest to Klein's (1946) original definition. Here the recipient does have a role, but a passive one. The projection is *onto* the object, who need not be affected by it. It is only within the subject's mind that the object of the projection takes on the function of being the subject's self – it is the projector who identifies with the recipient so that what happens to the recipient is experienced as though it were happening to the self. Thus, in the story of the students quoted previously, the female student need not accept the male student's projections or participate with him in the protest.

A more active role for the recipient is proposed in the third form of object involvement, favoured by Bion and his followers, whereby attributes are said to be projected *into* the object. In addition to any identification by the projector with the recipient, it is the recipient who, albeit generally unconsciously, identifies with what is projected and is induced to think, feel or behave differently. For example, the recipient may split, deny or act out aggression. Children seem particularly susceptible to accept and identify with projections. When parents deny a quality in themselves and project it into a child, they alter their behaviour towards that child. The parents thereby exert pressures, the result being a self-fulfilling prophecy as the child begins to behave in accordance with the projections.

For example: From the age of 12, a previously compliant girl displayed a cycle in which intense closeness to her parents alternated with violent temper outbursts towards them, followed by running away and dangerous promiscuity. This behaviour resulted in repeated admissions to institutions. When the mother disclosed that she had been repeatedly raped by her own father from the age of 12 and had been preoccupied about her daughter meeting the same fate, the girl's behaviour could be seen partly as an expression of her mother's unresolved and projected conflicts.

Although Klein did not explicitly describe this latter form of projective identification, I would agree with many current theorists that projection *into* an object represents the full expression, while projection *onto* an object is an incomplete form of projective identification. Similarly, although projection is often regarded as a more mature defence (Sandler, 1987), it is useful to think of it as an attempt at projective identification which fails to penetrate or attach to a recipient.

Acceptance of the interpersonal or intersubjective nature of projective identification helps to make sense of the idea that it can function as a form of *communication* as well as a *defence*.

Projective identification as communication

Projective identification can be a form of communication through which one person is made to experience or become aware of the emotions or other attributes of another. When it functions primarily as communication, projective identification is a normal and potentially positive process, perhaps best illustrated by a mother's ability to use the feelings that her newborn elicits in her to help her to make sense of that baby's needs. The process is difficult for both participants and observers to understand, because it takes place outside conscious awareness (although introspection may render it conscious). Just how the communication takes place is open to question. It seems to develop pre-verbally and is, therefore, largely independent of the overt meaning of words or actions. Sometimes authors seem to suggest that the process is inexplicable, but presumably subtle voice tones, gestures, etc. produce some resonance in the recipient. There are analogies in the way in which we are affected by music, so that I find it useful to think of projective identification as the music of interpersonal interactions. Perhaps the process depends in part on some shared experience. For example, the mother has been an infant herself, has been mothered, and, according to psychoanalytic theory, has unconscious memories of these experiences, which might be triggered by her experience with her baby.

Communication by projective identification is to be distinguished from coercive processes like indoctrination where a conscious attempt is made to influence. Hypnotic suggestion and charismatic leadership contain elements of both projective identification and conscious coercion, and are therefore of interest. Similarly, projective identification sheds a useful light on the observation made in communication theory by, for example, Watzlawick *et al* (1967) that we cannot "not communicate".

Projective identification as defence

When projecting emotions or qualities does not achieve communication, it may become a way of evacuating uncomfortable feelings for someone else to deal with, or a way of controlling the other person. When communication fails in this way, the process is likely to be destructive, and is in danger of becoming chronic and defensive. Again, it is clearly an interactive process, with the outcome depending on the state of mind of both parties.

> For example: I was feeling angry with a patient and could not understand why. My supervisor suggested that, through the patient's use of projective identification, I was being made to experience her anger. With this understanding, I could begin to recognise the communicative side of what was otherwise a chronically defensive pattern of interaction resulting in a potentially destructive evacuation. I was also less prone to feel angry towards the patient, illustrating how attention to projective identification can facilitate empathy.

Conversely, consider a mother whose state of mind prevents her from making sense of her baby's needs. She is not only unable to make use of her baby's distress as communication but also is likely to feel distressed, inadequate and even persecuted by the baby's behaviour. In a different state of mind, she may have been receptive to the communication, and responded to it in a way which could have enhanced her relationship with the child.

Ideally, when infants and children project feelings that they cannot manage into their parents, the parents are able to make use of the communication and respond to the child's needs in a way which renders the projected feelings safe. When adults project into children, however, the children are incapable of dealing constructively with the complex feelings with which they identify. As a result, the children are left to express a conflict denied by the parent in a way which is destructive for them and their family. Patterns of chronic, defensive use of projective identification within families are not uncommon, and are illustrated by the above examples of the promiscuous 12-year-old and the sex-role stereotyped couple. Because the child identifies with the parental projection, conflict can continue to be expressed in the absence of the individual who initiated it.

An attempt at synthesis

To summarise so far, projective identification can have positive or negative outcomes; can involve a variety of different mental mechanisms; allows a variable role for the recipient of the projection; and can have among its functions communication, control of others, and defensive avoidance. I have outlined these differences in a way which might give the impression of dealing with entirely separate processes; however, different examples have at least one important feature in common – a temporary or permanent breakdown of boundaries between individual psyches, so that processes which would be expected to be intrapsychic come to involve at least two individuals. Thus projective identification is said to be an important link between the intrapsychic and the interpersonal (Ogden, 1979).

The critical factor seems to be that actions or emotions appear to be experienced or processed by one individual even though they were initiated by or within another. The state of mind of the recipient will determine how and whether a particular projective identification is accepted. It might be that the projection is like a key, which to become a projective identification must find an individual with the right lock in the psyche. If not, it remains projection.

The function of projective identification between individuals is analogous to the action of hormone in the communication between cells. A cell disseminates a hormone more or less widely in its environment (the body). The hormone is 'ignored' by those cells which have no appropriate receptor for it, while it may or may not be received by those that do have receptors, depending on their state at the time. When the hormone attaches to a cell it triggers a process in it which is more or less specific to that hormone and that cell. The response of the cell might either be passive or active, either a simple reaction or involve the integration of a number of stimuli. Some types of hormone activate receptors on a wide variety of cells; others affect only a few specialised cells. The effect of the hormone varies from short-lived and minor modification in activity to a substantial, persistent change in function. Feedback mechanisms exist so that the receptor cell might in turn release a hormone or otherwise impinge upon the effector.

This analogy works only if the concept of projection *into* an object is accepted. But there is considerable disagreement about how literally projective identification is to be taken. At one extreme the process is conceptualised as taking place entirely in the fantasy world of the projector – "the experience is all mine and independent of you". At the other, the emotions and other qualities are said to pass from one to the other so that "you directly experience my feelings". In between is the view that one person unconsciously 'triggers' a reaction in another. Thus "you resonate to me or perhaps experience feelings similar to mine, but they are your own feelings, from your own inner world not mine". I find the second and third formulations more helpful.

Projective identification in general psychiatry

Projective identification is a focus of vigorous development within psychoanalysis, but the language used means that the work is not readily available to the general psychiatrist. As a result the concept has been under-exploited. I give three examples of areas in general psychiatry to which the concept can usefully be applied.

The clinical interview

Projective identification is pervasive in human interaction. Even when it fulfils a primarily defensive function, projective identification does communicate, provided the recipient is in a suitable state of mind to understand the communication. Psychiatrists can exploit this fact in their work by examining their own reactions. Typically in the course of a psychiatric interview both patient and psychiatrist will experience a variety of feelings such as anger, sadness, warmth, boredom, etc. Some of these feelings will be easily intelligible on the basis of anxiety about the interview and the context in which it occurs. Others will be brought to the interview by the participants on the basis of their remembered previous experience. A third category, which includes transference phenomena, is more difficult to qualify. It includes some feelings that are explicable without reference to the interaction between two parties. For example, a trainee may become angry with elderly female patients, more or less independently of those patients' behaviour. More often there will be feelings elicited in both psychiatrists and patient that is a product of the interaction between them and it is here that the concept of projective identification may be useful.

As trainees we were taught to recognise some of our own feelings as 'countertransference' and to appreciate the dangers of acting on them, but paid little attention to what we could learn from such reactions. Projective identification is not the only determinant of these reactions, but its recognition and understanding is important since it can cloud conscious perceptions and thus undermine management. Although care must be taken not to hold the patient responsible for all negative feelings evoked while seeing him/her, discerning projective intrusions from a patient can offer insight into his/her problem. Of course none of this is unique to psychiatry. For example, Balint (1964) discusses how general practitioners can learn from the effects that difficult patients have on them.

The psychiatric ward

The ward dealing with disturbed patients is a setting where intrapsychic boundaries can break down – not only between one patient and another but also between patients and staff and within the staff group. Projective identification is helpful in making sense of phenomena that can provoke anxiety and undermine therapeutic activity.

Stanton & Schwartz (1954), in their classic study using sociological methodology, demonstrated that staff disagreement was one of the most powerful determinants of excited behaviour among patients in the mental hospital ward. They postulated that the apparently unpredictable and disorganised behaviour of patients is not as chaotic as staff and patients experience it to be. Instead it has a structure which reflects, among other things, staff tension.

The maintenance of these unacknowledged structures is of course complex and multidetermined, but projective identification offers a possible explanation for how people's apparently independent behaviour is unconsciously influenced by the group or institution. Psychiatric wards are particularly susceptible to such influences because of the distorted and ambiguous communication which is a part of severe psychiatric disturbance. Being alert to the effects of projective identification in the ward can help the psychiatrist to make better sense of the interaction between patient behaviour and staff anxieties and dysfunction. There is a case for engaging outsiders to consult to groups of staff who work with disturbed patients in psychiatric settings, at least until the insights have become part of the ward culture.

Working with families

Until recently the family therapy literature has emphasised the difference between various approaches to working with families. It is often argued that integration between psychodynamic and systemic approaches is impossible since the theories are incompatible. Family therapists, disenchanted with what they see as limited success of psychoanalytic understanding in changing behaviour, have under-rated the relevance of intrapsychic phenomena to their work. Projective identification offers an additional and useful way of making sense of concepts such as enmeshment, scapegoating and triangulation – concepts that family therapists often accept at face value. The undercurrents that maintain a family in a dysfunctional state might be better understood if attention is given to the way in which the intrapsychic state of an individual can affect what happens between individuals. Some of the clinical examples quoted above should support this contention.

A technique that is frequently used in family therapy is for a consultant to observe the therapist and family from outside the room by camera or other means. It is found that while the therapist is drawn into the family system or way of relating, the consultant is able to remain relatively objective about the interactions. The consultant is then able to make use of the reactions of the therapist to increase the overall understanding of a family. Compare this process with the use in individual therapy of countertransference as a tool to make sense of projective identificatory communication.

Conclusions

Projective identification is one of many ways of thinking about the influence we have on one another outside conscious awareness. The various phenomena described do not fit a single definition, but they do form a recognisable group of psychic functions which play an important part in psychiatry, psychotherapy and social psychology. Projective identification offers an approach to their understanding which I believe is compatible with the practice of psychiatry in a hospital. Furthermore, this account perhaps offers something of a bridge between psychoanalysis and psychosocial/systemic approaches, a communication which might allow each to inform and be informed by the other.

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The TAPS Project. 3: Predicting the Community Costs of Closing Psychiatric Hospitals

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The planning of long-term care in the community as an alternative to in-patient care requires accurate information on the likely expense of altering the balance of provision. Unfortunately, as very few long-stay psychiatric hospitals have yet closed, the planning of these resource requirements has had to proceed in a vacuum. By examining the costs of community reprovision for the first 136 people to leave Claybury and Friern Hospitals, a prediction equation has been estimated from existing data which links the hospital-assessed characteristics (including psychiatric symptoms and behavioural problems) of these people to the subsequent cost of community care. About a third of the observed variation in these costs can be explained statistically by these 'baseline' characteristics. However, the first cohorts exhibit fewer behavioural problems and other symptoms of mental illness, they have been in hospital for shorter lengths of time, and they are younger. The prediction equation for the leavers is thus used to extrapolate community costs for those hospital residents yet to leave. It is found that community costs are *lower* than hospital costs, not just for the first cohorts of leavers, but for the full populations of the two hospitals scheduled to close.

One of the key questions to be asked of the government's policy of substituting community care for hospital residence for people requiring long-term care is its cost. Hospital run-down and closure generate large savings, but are they large enough to fund the replacement community provision?

The answer to this straightforward question has proved elusive. The costs of community provision can only be measured directly for those people who have *already* moved from hospital, and then only with difficulty. Moreover, it is almost always the patients with fewer or less severe needs who are the first to leave hospital, and it is likely that their care will cost less than the care of the more dependent patients left behind. It is invalid to generalise from the experiences of the first cohorts of movers to full hospital populations unless one can be sure that the two groups are identical in every relevant respect or unless differences between the groups can be incorporated into the cost predictions.

In this paper we report an attempt to predict the costs of community reprovision for the full long-stay populations of two large psychiatric hospitals. Given comprehensive costings for a sample of former residents and detailed clinical, demographic and personal data on the full hospital populations (or at least those parts of the populations relevant for the present predictions), we examine the statistical relationships between community costs and the characteristics of movers when they were in hospital. The observed relationships are then used to predict the costs of psychiatric reprovision for those people who have yet to leave hospital.

Extrapolations of this kind are replete with difficulties, being dependent on both the quality of the data and the robustness of the statistical analyses. We do not pretend that the data and the techniques employed here are perfect, but we would argue that the present extrapolations are based on the most reliable estimates presently available. As yet there is nothing better to inform the debate. On even the most cautious assumptions we arrive at estimates for future expenditure requirements which are less pessimistic than most of the critical rhetoric that surrounds this controversial area.

This paper is *not* concerned with the impact of community-care policies on acute mental health services, although this is a pressing topic for research.

The provision of community care

In 1983 North East Thames Regional Health Authority (NETRHA) announced plans for the partial run-down or closure of all its long-stay psychiatric hospitals. Friern and Claybury Hospitals in north London, the largest in the region, were the first to begin the run-down and were to be closed within ten years. Bed numbers had in fact been falling for many decades. In the years immediately preceding the announcement, most of the decline was accounted for by deaths. During 1980–85, for example, an average of 23 long-stay Friern patients died each year, and 40 were discharged, but between 40 and 50 'new long-stay' accumulated each year. Under the newly introduced 'dowry' arrangements, an annual sum of money equal to the average

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revenue costs per patient of running each hospital was to be transferred from the hospital budget to the district receiving a hospital leaver whenever this resulted in the permanent closure of a bed. (Setting the dowry at average hospital revenue cost is more generous to community services than the systems in operation in many other English health regions, as we note later.) In 1988 this transfer payment was between £16 000 and £17 000 per annum. Long-stay patients, defined as those with at least 12 months of continuous hospital residence, were covered by these dowry arrangements if a new place in the community was created for them. Where health districts do not provide the service, some or all of the dowry money can be passed to local-authority social-services departments or voluntary organisations.

The initial plan for Claybury was complete closure by 1993, with the site perhaps sold off for private housing or shopping developments if planning consent could be obtained. The anticipated future site value was to be converted into capital funds for new community facilities, particularly small staffed group homes and day facilities, and made available to district health authorities (DHAs) on presentation of acceptable reprovision plans. It was never intended that Friern should close completely. It was envisaged that Haringey DHA could convert part of the listed site into a mental-health facility with both acute and some sheltered longer-stay care. However, it was initially expected that all long-stay patients would have been 'decanted' into community accommodation by 1993. In the event, and in line with experience nationally, plans for the run-down of Claybury are slightly behind schedule. It is now expected Claybury will close in 1994. The closure date for Friern remains as 1993. By June 1989 a total of 146 long-stay patients had left Claybury and 112 had left Friern, many of them under the reprovision arrangements which carried dowry transfers. Most of these people moved to accommodation within London, spread across eight DHAs and nine local authorities.

NETRHA commissioned research to evaluate the 'psychiatric reprovision' plans. This research programme has been gathering data since before the first reprovision patients moved to the community in 1985, and has covered: detailed assessments of in-patients; follow-up assessments of leavers one and two years after discharge; equivalent follow-up assessments of a sample of 'stayers' matched on age, sex, hospital, duration of hospital stay, case-note diagnosis and social behaviour; and costing of both hospital and community services (Team for Assessment of Psychiatric Services (TAPS), 1988). A randomised design, whether for hospitals or patients, was out of the question, and it must be recognised that, once the decision had been taken to close the hospitals, the care provided within them began to change as staff and money moved, as maintenance expenditure was cut, and as wards closed as the first cohorts of patients left. In fact, it would appear that the 1986–87 revenue costs for in-patient care per day at Friern and Claybury (£49.92 and £42.12 respectively) compared well with the average cost per in-patient day for all mental hospitals in ten of the English health regions (£44.05) (author's calculation). Whether the long-stay populations of the two study hospitals are typical of the national situation is impossible to say, given available national data collections.

This paper focuses on patients who have been in continuous residence for at least a year, and who, if over 65 years old, do not have a current diagnosis of dementia. Baseline information for all such patients resident in both hospitals was collected, ranging over a number of dimensions, including mental health status, using the Present State Examination (PSE) and the Social Behaviour Schedule (SBS); the Physical Health Index (PHI); patient personal and historical data; patient attitudes; information on patients' social networks, using the Social Network Schedule (SNS); and an assessment of living environments (Freeman *et al*, 1965; Wing & Brown, 1970; Hall, 1980; Abrahamson & Brenner, 1982; Dunn *et al*, 1990; Leff *et al*, 1990). Altogether, including the 'new long-stay' patients who had accumulated in the hospitals since the study began, baseline information was assembled on 964 patients.

Costing community care

There are four simple rules to observe when costing health or social care services (Knapp & Beecham, 1990*a*). Firstly, the costings need to be comprehensive. They should range across all relevant service components of a 'package' of care. Secondly, the costings will reveal probably quite wide variations between different patients, facilities, areas, and so on. These variations must not be glossed over; they provide a wealth of information which, if handled properly, offers valuable policy insights. Thirdly, cost comparisons between individuals, facilities, and areas should be undertaken cautiously. Only like-with-like comparisons have validity. Finally, cost findings should ideally be integrated with outcome or effectiveness findings. Certainly, we would never advocate sole reliance on cost in the formulation of new policies or clinical practices, except under very special and, generally, rare circumstances. Equally, we would not be happy to find policies built on outcome evaluations which paid no heed to costs.

These four rules have guided the costing methodology employed in the Claybury and Friern study, based on the Client Service Receipt Interview (CSRI) developed at the PSSRU, University of Kent at Canterbury (unpublished). The CSRI is completed one year after discharge from hospital. The costs of community care are based on retrospective accounts of service utilisation, accommodation descriptions and histories, staffing arrangements, and social security and other income receipts. They are defined so as to cover every service used, and priced to reflect the long-term marginal costs of care, including the opportunity costs of capital. (Consequently, the costs for these first cohorts are not artificially deflated just because these early leavers can be moved to existing residential accommodation or day centres). All costs are based on London salary weightings and land prices, both for hospital and community services. Outside London absolute costs would have been lower in 1986–87 than for this sample of people, but differences in cost between patients and between hospital and community care, which are the focus of this paper, have wider currency. The costing reflects service utilisation and living arrangements in the 12th month after leaving hospital, although adjusting for regularly but infrequently used services such as occasional short-term in-patient stays

TABLE I
The costs of psychiatric reprovision for 136 long-stay patients

Components of total cost	*Percentage using the service*	*Average weekly cost: £*		*Percentage contribution to total*
		users	*full sample*	
Accommodation and living expenses	100	209	209	77.4
Hospital out-patient	21	9	2	0.7
Hospital day care	32	48	15	5.4
Other day care	38	33	12	4.5
Education services	10	33	3	1.2
Police	4	5	0	0.1
General practitioners	72	2	1	0.5
Injections	14	6	1	0.3
Nursing services	30	4	1	0.5
Psychiatrist	54	2	1	0.5
Social worker	42	23	10	3.6
Miscellaneous professionals[1]	58	3	2	0.6
Travel	32	2	0	0.2
Volunteer inputs	4	17	1	0.3
Hospital in-patient stays	20	61	12	4.4

[1] Chiropodist, optician, dentist, pharmacist, occupational therapist, physiotherapy, psychologist.
Total cost per person per week (1986–87 prices): average (mean) £270, standard deviation £121, minimum £47, maximum £568

throughout the full first year in the community. Informal care was provided to only *one* client on a significant and regular basis. This has not been costed. Everyone else received formal care.

The community costs of 136 of the 161 leavers in the first two years of the study are summarised in Table I, at 1986–87 prices. (The leavers for whom it was not possible to calculate costs were not significantly different from the sample of 136 on any of the key variables.) Mean cost per person per week was £270, although varying by a factor of as much as 12 between patients with the lowest and highest costs. The majority of the reprovision cost is accounted for by accommodation and day-to-day living expenses (more than three-quarters of the total). Further details of the methodology and the components of cost are reported by Beecham & Knapp (1989).

The patient outcomes of reprovision

Based on these compehensive costings, we examined the variations between patients. It is not the purpose of this paper to detail the findings of the outcome evaluation, but it is crucial that we do not view costs in a vacuum. Anderson *et al* (unpublished TAPS paper) found, for the first 161 leavers all moving from hospital in the first two years of the reprovision policy, no adverse effects of relocation on clinical and social outcomes. For example, none of the aggregate or subscale scores on the PSE, the SBS or the SNS showed statistically significant differences over time (comparing assessments in hospital and community) between leavers and their matched stayers. However, statistically significant differences between leavers and their 'matches' over time were revealed in terms of positive attitudes to present accommodation and a reduction in the 'restrictiveness' of the environment (Wing & Brown, 1970). The leavers considered medication of less help than did the matched stayers. It is important to keep these encouraging patient outcome findings in mind when looking at the costs.

Explaining community cost differences

The mean cost per person per week disguises a wide range. Lowest cost is £47 per week for a man living independently in a one-bedroom flat he shares with a friend. Although there are no staff formally attached to the accommodation, a field social worker visits every week and the client has monthly out-patient appointments and appointments with a general practitioner (GP). At the other end of the cost range (£568 per week), there is a woman living in a staffed group home managed by the health authority. She has a high level of support from her GP, social worker and a psychiatrist, but also spent three months in Friern Hospital during her 'reprovision period'.

What explains such wide variations in cost? Clearly, mental and physical health is likely to be relevant. It might be the case, for example, that higher costs are associated with better health (and other facets of individual well-being) through service use and effectiveness. Local policies and services should have some influence. It may be the case that non-statutory facilities are less expensive, other things being equal, than their public-sector counterparts. Each of these hypothesised cost-raising factors will be examined later in the NETRHA research programme (compare Knapp & Beecham (1990*b*) for a recent demonstration programme). For the moment our objective is to use the baseline (hospital) characteristics of leavers to explain the observed variations in community costs. Only information available before each patient left

TABLE II

The estimated reprovision cost prediction equation (dependent variable = cost per week at 1986–87 prices)

Explanatory variables[1]	*Coefficient*	*Significance*[2]
Constant/intercept term	138.51	0.000
MALE	45.68	0.066
MALE × TOTNAM	−3.28	0.007
LIFE	597.10	0.019
LIFE squared	−968.54	0.024
STAY squared	0.002	0.016
STAY × AGE	−0.01	0.067
STAY × TTMH	0.04	0.007
SBSTOT	11.56	0.003
PSENEG × AGE	0.75	0.006
PSENEG × TOTTIM	−0.092	0.090

$R^2 = 0.383$, $\overline{R}^2 = 0.326$, $F = 6.705$ (significance 0.000), $n = 119$[3]

1. The mnemonics are explained in Table III.
2 Significance of *t*-test of individual coefficients.
3. For 17 of the 136 subjects we did not have enough information to measure the variable TOTNAM.

hospital was entertained as an acceptable explanatory variable for these purposes.

Taking cost per week as the dependent variable and the full range of patient data collected in the TAPS interviews as the set of potential explanatory factors, we used ordinary least-squares multiple-regression analysis to tease out the prediction relationship. (Costs of community care were found to be normally distributed around the mean, making this analytical approach appropriate.) Clinical and other characteristics of patients were introduced into the regression equations both singly and in additive and multiplicative combinations (including higher powers) in an attempt to maximise the percentage of variance explained, although with an eye on the statistical significance of individual regression coefficients and the interpretability of results. Some of the potential explanatory variables are intercorrelated. This multicollinearity does not affect the predictive power of the equation or the validity of the extrapolations reported later, but it can make it difficult to disentangle the relative importance of the individual variables. For this reason we took a fairly generous cut-off level of significance ($P < 0.125$) in the analyses.

The 'best' prediction equation, by these conventional criteria of statistical significance, parsimony and interpretability, is detailed in Table II. (Note that the 'next best' regression equations are little different from this one – one or two variables drop out to be replaced by similar indicators of patient well-being and background, with the overall explanatory power of the equation slightly reduced, but the extrapolations to the whole hospital populations which we report later are unchanged.) The mnemonics used in Table II are explained in Table III, where we also report the basic statistical properties for these variables for both the sample of leavers for whom we had cost data and for all patients in the two hospitals who have been in continuous residence for at least a year and who, if over 65 years old, do not have a current diagnosis of dementia. Overall, the equation explains 38% of the observed cost variation. This is a high level of statistical explanation given that we have been using only baseline information in the equation. It implies that comprehensive hospital assessments of patients' needs can predict more than a third of the subsequent variation in community costs. When we come to introduce community information (e.g. patient needs and outcomes, local circumstances, location) into the cost analyses – as we will be doing in later research – we should be able to explain a much higher proportion of the cost variation.

The equation in Table II offers some interesting perspectives on those characteristics of hospital residents that are correlated with the subsequent need for support in the community and hence the costs of psychiatric reprovision. Men on average cost £20 more per week than women, even after standardising for mental health state and all the other factors in the equation. We interpret this as implying that in the placement of long-stay patients in community accommodation and in the planning and delivery of support services it is assumed that males are less capable of self-care, and are either directed towards more highly staffed accommodation or offered more closely supportive (more costly) care. As with many of the modelled effects on cost, the influence of gender is not simply linear,

TABLE III

The hospital characteristics associated with reprovision cost (£ per patient per week)

Mnemonic variable		*Sample of costed leavers*			*Full hospital populations*		
		mean	*s.d.*	n	*mean*	*s.d.*	n
MALE	Indicator variable = 1 if male; 0 if female	0.52	0.50	136	0.57	0.50	964
AGE	Age at time of hospital assessment	54.96	14.22	136	58.24	15.98	963
STAY	Current length of stay: months	154.78	171.04	136	222.09	211.90	964
TTMH	Total previous time in hospital: months	52.99	89.10	136	42.64	78.44	964
TOTTIM	Total time ever in psychiatric hospital: months	207.83	178.24	136	265.09	209.39	964
LIFE	Proportion of life spent in hospital	0.30	0.22	136	0.35	0.24	963
TOTNAM	Total number of named social contacts	11.61	9.33	120	8.68	6.94	604
SBSTOT	Total score on Social Behaviour Schedule	3.44	2.90	136	5.50	3.44	963
PSENEG	Score on Present State Examination for negative symptoms	1.02	1.08	133	1.25	1.27	951

nor is it independent of all other cost-raising factors. In this case, gender and the size of a patient's social network in hospital are inter-related (see below).

The effect of patient age is mediated through two other variables – continuous length of stay in hospital before reprovision discharge date and the negative symptoms score on the PSE (see below) – although they do not alter the general conclusion that the care of older patients is less costly. These older patients are probably perceived as being less demanding insofar as they are not expected to need, say, employment programmes or further education. (The receipt of any day care or day activities was significantly lower ($P<0.1$) for people aged 70 or more.) Among those patients whose original diagnosis was schizophrenia, it is likely that older people and those spending longer in psychiatric hospital will have reached a stage where negative symptoms are more predominant than positive symptoms. Although they are less likely to have moved from hospital in the first two cohorts, the older patients in Friern and Claybury, it appears, can be accommodated in the community at less cost.

In a number of ways a patient's history of in-patient care exerts an influence on the later costs of community reprovision. The current length of stay, the total time ever spent in hospital, and the proportion of a patient's life spent as an in-patient all have statistically significant influences. (Recall the intercorrelations between some of these variables which can make it harder to disentangle the relative cost-raising contributions of individual variables.) In some cases the influence is modest, as with the total time ever in hospital (all in-patient stays added together), or the total previous time (excluding current stay) in hospital. Length of stay before discharge under the run-down programme has no real influence on cost until it exceeds about ten years. There is a curvilinear relationship between cost and length of stay (and mediated through some other factors) which suggests that, beyond this ten-year point, a longer stay increases costs. The effect is quite marked and will be influential in our subsequent extrapolations since the mean length of stay is 19 years for the long-stay population under study. What this result suggests is that the effects of prolonged institutionalisation as measured by the receipt of more costly reprovision packages, are noticeably greater only after some years but are then quite marked.

The final measure of in-patient experience is the proportion of each patient's life spent in psychiatric hospitals. This ranges from 1% to 99% around a population median of 34%. Community costs are generally higher for those people who have spent higher proportions of their lives in hospital, another empirical association interpretable as an institutionalisation effect. In fact, 53% of the whole hospital populations (508 people) have spent more than 30% of their lives in hospital. Comparison of the variables in the cost function shows this group to have, on average, a slightly smaller social network (1.1) and slightly higher scores on the PSE negative symptoms (0.22) and total SBS score (0.48).

The other significant influences on the costs of reprovision all relate to an in-patient's well-being, including his/her clinical state, at the time of the hospital-based assessment. This assessment is not simply an indication of, say, psychiatric symptoms on a single day, but identifies general symptoms over a longer period (one month for the PSE, SBS and SNS, and one year for the PHI). Three separate indicators appear in the final analyses – the PSE negative-symptoms score, the total score on the SBS and one of the measures of social contacts as measured in part of the SNS. In-patients with higher scores for negative symptoms were socially withdrawn, showed flattening of affect, and displayed poverty of speech. By repute, these people are the hardest to place in the community. We have now found that they are likely to be receiving more costly service packages in the community. This effect of the PSE score was attenuated for older patients and those with longer spans of in-patient residence, but still with a positive association with cost. Higher scores on the SBS indicate greater staff-reported ratings of abnormal behaviour (Wykes *et al*, 1982). The effect of SBS scores on cost is as expected: patients with more social behaviour problems are supported in the community at greater cost than those with fewer such problems, other things being equal. Finally, patients with a larger number of named social contacts when assessed in hospital, that is with wider social networks, cost less to reprovide for than their more withdrawn peers, both because of the association with general psychiatric status and because, being more communicative and gregarious, they make fewer demands on care staff and support workers. In fact, this result is confined only to males, and a female leaver's social network exerts no influence on cost. Disaggregated scores on the SNS, for example, the percentage of contacts listed as close friends, were not significantly related to costs.

A number of baseline characteristics proved to have no effect on community cost when analysed within a multivariate context. These included marital status, ethnic origins, patient attitudes, physical health, original psychiatric diagnosis, reasons for in-patient admission, number of medications, and hospital of residence (Claybury or Friern).

The prediction of later reprovision costs from circumstances and characteristics rated in hospital has revealed a closer causal linkage than we had expected to find. Many of the links are far from straightforward in that a variety of non-linear variables most successfully captures the underlying relationships. Encouraged by the reasonable degree of (multiple) correlation between baseline data and community costs we can now extrapolate these costs of reprovision for all long-stay patients.

Extrapolations of cost to full populations

Health-authority managers need to have some idea of the future resource implications of their hospital run-down and closure plans. Transfers of funds from hospital coffers to community budgets are intended to be the mainstay of the new psychiatric services for chronic populations, bolstered by as many and as generous social security benefits as the system can be persuaded to provide. Therefore, health-service planners (and, if recently announced government plans materialise, local-authority social-service planners) need information not just on the costs of today's leavers but accurate indications of the costs of supporting the whole hospital population once 'decanted' (or the relevant subpopulation likely to move into the community). Only with such data can they begin to formulate long-term plans for accommodation, day support and peripatetic professional staff, and also enter into informed discussions with other agencies as to the co-ordination of policies and services.

Every time a costing is produced for former long-stay hospital patients, the riposte is heard that future hospital leavers will cost much more. This riposte has undoubted face validity, but to date it has proved difficult to test its veracity, and it has certainly been impossible to say just how much more expensive their service packages will prove to be. There are perhaps three reasons for expecting higher future costs. Firstly, the early movers in most hospital rundown programmes are less dependent, with fewer symptoms of mental illness, than those who remain behind to move in later cohorts. Secondly, it is commonly the case that new capital projects are not needed for the first leavers because existing day and residential facilities are as yet not operating at full capacity. Thirdly, although often overlooked, it is possible that the supply price of certain types or grades of staff may rise with the growth of demand for them in the community. It is difficult to assess the burden on families and informal care networks, but increased amounts of support for carers will be necessary. Of course, the rundown of hospitals will release a number of medical and nursing staff for redeployment elsewhere, but it is by no means clear, nor necessarily desirable, that the two effects should cancel out.

The third of these reasons for expecting later cohorts to be more expensive may have only a limited inflationary effect, and anyway is virtually impossible to build into cost predictions without a study of professional labour markets. We have made a modest attempt to recognise the likely deviation between current and future input price levels by placing a price on the services of volunteer staff on the assumption that there is not an unlimited supply of such people. The second reason for greater future costs – the need for major capital spending only for later cohorts – is, in research terms, a misspecification error: it relates to flow of expenditure but not cost. If services are costed at long-term marginal cost from the outset, as in this study, the argument does not hold. Capital costs are already set at their long-term replacement values. We are left with the differential characteristics of leavers and stayers as the principal source of future cost inflation.

Employing the estimated cost equation for the 119 leavers for whom we had complete data, and the baseline descriptions for the study populations of 450 Claybury and 514 Friern residents, we predicted the cost of community living and support for every patient targeted to move.

The validity of these predictions depends on a number of things. Firstly, the prediction equation must be robust. We would certainly prefer, from the perspective of the robustness of predictions, to have a cost equation with a higher multiple correlation coefficient, but the results (Table II) are still highly significant. (Incidentally, a much higher correlation could have the depressing interpretation that community-care services were shaped predominantly by historical hospital characteristics and responded less to potential changes in characteristics or needs thereafter.) For the purposes of prediction and comparison with hospitals, it is also worth noting that the confidence intervals around the regression equation are fairly tight. For example, median predicted average cost is approximately £296. The 95% confidence intervals around this median, taking each of the predictors in turn (and setting all other variables to their median values), are all within £20 either side of this predicted cost. Thus, with 95% confidence we can expect median predicted cost to be within the fairly narrow range £276–316. Slightly different ranges apply at different costs.

Secondly, there must be no correlation between the cost-function residuals and any variables (within or outside the function itself) which predict which hospital in-patients moved to the community. Checking of all available 'baseline' information collected in hospital (139 variables in all) revealed no such sample-selection bias. Thirdly, even though we expect the early cohorts of leavers to be less dependent and to have fewer symptoms than those who currently remain in hospital, the validity of the extrapolations depends on the differences being not too great. In particular, the observed span of values on each of the cost-influencing variables listed in Table II for the sample of 119 people covered by the cost analyses needs to be not greatly dissimilar to the corresponding span for the full population, even though we know that mean values are

TABLE IV

Ranges of variable values, for sample of costed leavers and hospital populations

Mnemonic	*variable*	*Sample of costed leavers*		*Full hospital populations*	
		min.	*max.*	*min.*	*max.*
AGE	Age at time of hospital assessment	23	85	19	101
STAY	Current length of stay: months	12	711	4	863
TTMH	Total previous time in hospital: months	0	533	0	622
TOTTIM	Total time ever in psychiatric hospital: months	13	711	12	936
LIFE	Proportion of life spent in hospital	0.01	0.80	0.01	0.99
TOTNAM	Total number of named social contacts	0	47	0	47
SBSTOT	Total score on Social Behaviour Schedule	0	12	0	16
PSENEG	Score on Present State Examination for negative symptoms	0	4	0	6

different for some variables. From Table IV it can be seen that the ranges of the scores on the influential variables do not overlap completely, but are not so far apart as to cause difficulties with extrapolation. Looking at Tables III and IV we have clear evidence of the selective rehabilitation of patients – those who are more dependent remain behind. (This is not a comprehensive account of differences between the two groups since we focus on only the cost-influencing variables.) However, the averages disguise the fact that on many of the indicators relevant for the prediction of costs the leavers and 'stayers' are little different.

Fourthly, we have to make some adjustment for missing values if these force us to drop unrepresentative groups of patients from the predictions. In the event, only one of the variables in the equation generates a problem of this kind – the total number of named social contacts recorded within the SNS ('TOTNAM'). This information was obtained in interviews with patients themselves, and those who could or would not communicate had to be credited with 'missing information'. The problem is that these 'non-communicators' were much less likely to have left hospital in the early cohorts and are typical of neither the sample of leavers for whom we were able to measure costs nor the full hospital populations. Their scores on some of the key variables in the cost predictions (and hence the extrapolations) are significantly different from the scores registered by people who were able or willing to communicate information on their social networks. (They have, for example, spent longer in hospital since their most recent admissions and lived more of their lives in psychiatric hospitals; they score higher in terms of both PSE negative symptoms and SBS.) It would, therefore, be dangerous to drop these 'non-communicators' from the extrapolations, and we substituted a number of alternative scores for the missing values of the variable TOTNAM. From Table II we note that lower scores on TOTNAM produce higher community costs for males and have no effect for females. Given the nature of the variable TOTNAM it is reasonable to conjecture that, on average, 'non-communicators' have smaller social networks than other in-patients. But if we set TOTNAM to its smallest possible value (zero), costs will be £56 higher per week (on average) than if it is set to its maximum observed value. Set at the more appropriate estimate (based on the population mean for males of 8.658) costs will be merely £6 per week greater. In the extrapolations which follow and which are tabulated, we have set TOTNAM to its (male) population mean for the 'non-communicators'.

The results of our prediction are summarised in Table V. The predicted reprovision costs for those in-patients who had not moved from hospital in time to be included in the cost-equation analyses are approximately £61 more per week than the current mean of £270 for all leavers to date. The observed range of predicted costs for later movers (£119–882) when compared with the range for leavers to date (£47–568) clearly demonstrates from a cost perspective how the 'dependency' characteristics of those people currently in hospital will not only require more support *on average* in the community, but that there will also be a need for a more varied response from community reprovision services.

Comparisons with hospital costs

The mean community cost per person per week at 1986–87 prices is £321 for the full hospital populations defined by the study criteria. This is 19% above the reprovision costs measured for the sample of leavers to date. How does this compare with the costs of running the two hospitals?

The revenue costs, estimated capital costs (sale values with annual adjustments) and indirect costs for Claybury and Friern are given in Table VI for the last four years. (Note that sensitivity analyses taking different site valuations reveal no changes to the conclusions of this paper with regard to hospital–community cost differences; site-value inflation or deflation will also affect community facility costs.) These have been revalued to 1986–87 prices for the purposes of comparison. Table VI also gives average numbers of in-patients and beds for these same years. The impact of the reprovision policy introduced in 1985 can be seen from the changes in the numbers of available beds – a decline of 17% in Claybury and 3% in Friern. The effect on the inflation-adjusted costs is evident from Table VI. As hospitals run down towards closure, resettling the less dependent patients first, so the 'average dependency' of

TABLE V
Reprovision cost predictions and extrapolations for full hospital populations

Sample or population	*Average cost per patient per week: £ at 1986–87 prices* *mean*	*s.d.*	*min.*	*max.*	n
Costed leavers					
actual cost	270.35	121.06	46.92	568.48	136
predicted cost[1]	268.77	76.50	38.70	504.94	133
All leavers, predicted cost[2]	267.55	73.39	38.70	504.94	161
Stayers (to date), predicted cost[2]	331.54	87.60	119.43	881.69	789
Population (total), predicted cost[2]	320.69	88.64	38.70	881.69	950

1. Predicted cost differs from actual cost at mean because the cost-prediction equation was based on only 119 leavers.
2 Predictions based on assumption that TOTNAM set equal to male population mean value of 8.658 for those males for whom this information is missing.

Table VI

Claybury and Friern Hospital costs (£ per patient per week) and in-patient numbers

Cost per in-patient week[1]	*1984–85*	*1985–86*	*1986–87*	*1987–88*
Claybury Hospital				
revenue costs[3]	265.82	276.20	294.84	316.81
capital costs	48.43	52.19	54.81	55.98
indirect cost[2]	11.89	12.04	12.32	11.02
Total cost	326.14	340.43	361.97	383.81
Average no. in-patient cases[3]	1425	1272	1265	–
Average no. beds available[3]	1036	945	907	865
Friern Hospital				
revenue costs	312.58	304.30	394.44	376.12
capital cost	40.91	41.07	44.45	46.47
indirect cost	12.59	12.46	13.14	11.45
Total cost	366.08	357.83	452.03	434.04
Average no. in-patient cases	1373	1324	1447	–
Average no. beds available	890	878	848	867

1. All costs reflated to 1986–87 prices.
2. Calculated as £7.90 social security receipt plus 1.5% of revenue costs (Knapp *et al*, 1989).
3. Data from NETRHA annual accounts.

those remaining behind increases, with a predictable impact on costs. Cuts in hospital maintenance expenditure over the last few years in anticipation of eventual closure and possibly too the difficulties of retaining and recruiting staff may in fact have dampened the cost inflation, as well as possibly reducing the quality of the environment, but the increases are still marked. Similar inflation has been experienced in virtually all England's psychiatric hospitals.

The most appropriate year for the costs of the two hospitals for comparison with the community cost predictions is then 1986–87, since this was the year in which the number of long-stay beds in the two hospitals began to decline under the new regional policy of community reprovision. Comparing Tables V and VI it can be seen that the direct revenue costs (on which, in the North East Thames Region, are based the dowry transfers from hospital budgets to district health authorities) in Claybury are insufficient in themselves to wholly finance the community services that we are predicting will be needed by the full hospital populations, the difference being some 9%. In Friern, direct revenue costs are actually *larger* than the expected community costs.

However, the calculated reprovision costs include all services and all appropriate capital expenditures, and it is more relevant to make a cost comparison that also includes the capital and indirect elements of hospital costs. Against this benchmark, the predicted future community costs are lower than both hospitals' totals. This hospital total is not a notional figure. It is the *real* saving from moving the two hospitals to their respective closure targets, for it includes: the direct revenue costs of running the two hospitals, which will all be saved once the run-down has been completed, the capital valuations of the two sites which would be realised were they to have been sold (under a number of different assumptions as to site values, without altering the conclusion), and the indirect costs, such as the social-security payments to in-patients which allow the purchase of cigarettes and confectionary, and the costs of field social workers and volunteers, which will be saved or redeployed to provide equivalent services organised within, say, community settings. Even if we adjust the hospital costs to take account of the fact that present (and future) reprovision patients generally live on wards whose costs are less than the hospital average (because acute and psychogeriatric wards are more highly staffed), projected costs of community care are still much lower (at Friern) or marginally lower (at Claybury) than hospital costs.

Conclusions

Our finding that the costs of community reprovision packages for the populations of Claybury and Friern once relocated are no larger and probably smaller than their present hospital residence costs is, we believe, robust and significant. It has policy implications, and because we have been able, for the first time in Britain, to make informed conjectures about the financial consequences of hospital-closure policies, the implications carry particular interest. Of course, there are other policy questions which can only be answered in later stages of this research programme. What, for example, are the community correlates of the observed cost variations? To what extent are service packages and costs responding to patient needs? Does higher expenditure ensure superior clinical or social outcomes? Will the outcomes recorded to date be maintained for these early leavers as they continue their lives in the community? What are the longer-term costs of reprovision, given that the costs examined in this study are describing the situation after only 12 months? The answers to these questions must wait, but what are we to conclude from the findings to date?

If we first recall that there were no differences in clinical or social outcomes between leavers and the matched sample of stayers, and in terms of patient attitudes and environmental restrictions the leavers appeared to be faring better, we can hardly conclude that community care is being offered 'on the cheap'. Quality of life is at least as good in the community as in hospital for those people who have moved to date. Our cost equation could, therefore, be interpreted as indicating the cost of providing *at worst* acceptable quality services and equivalent quality of life to that experienced in hospital. Indeed, the financial transfer arrangements in NETRHA (transferring up to the average revenue cost of hospital to districts receiving reprovision patients) may actually encourage a *more* costly community response than is the case in regions where smaller amounts are transferred.

Once the hospitals have closed, and in fact rather earlier than that (see below), the savings will meet most of the costs of community services. But this does not mean that all is well (Knapp, 1990). The first thing to note is that the full saving from running down hospitals will not be reaped until full closure has been achieved. In fact, hospital-revenue cost savings need not lag much behind patient moves at the current rate of run-down across England. A study of costs of psychiatric hospitals in 10 of the 14 English health regions suggests that perhaps as much as 81% of average revenue costs can be saved within a year of each patient's move to a community placement which results in a permanent bed closure in hospital (Knapp *et al*, 1991). This rate of cost decline is faster than that claimed by hospitals, and requires the relocation of patients within hospital to allow ward closures, which is generally *not* desirable on clinical or social grounds. But it must be remembered that there is the danger that transfers of dowry payments to fund community reprovision can leave hospital in-patient services with insufficient resources to function at an acceptable level. Given the parallel needs for adequate and usually advance funding to establish new community services, it is clearly imperative to have some 'double funding' during the transition period.

In contrast to the arrangements in some regions, the dowry transfers within NETRHA are comparatively generous. However, even these payments do not compensate all agencies involved in community care. It is not a question of the *adequacy* of the payment but its *disbursement*. Family-practitioner services may not provide one of the largest inputs to reprovision packages, but the burden of medical care can fall quite heavily on individual GPs. Voluntary organisations can also be making important contributions. In neither case will there necessarily be any financial compensation from the dowry transfers. As more and more psychiatric patients move from hospital, the lack of adequate compensation could create disincentives for these individuals and agencies. The government's proposed reliance on case management in community care will be crucial for the co-ordination of services, but in itself may not overcome such disincentives.

The abilities of people with mental health problems, or any other long-term needs, to adjust to life 'in the community' after 10 or 20 years in hospital may be limited, and, no matter how good the hospital-based rehabilitation processes, the development of self-care skills and social competencies could take months or years. The cost and patient outcome findings reported here relate to the first 12 months after discharge. In second and subsequent years the needs for mental health care and support in the community could well alter. This is on the agenda for future research.

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The Assessment of Psychiatric Disability in the Community
A Comparison of Clinical, Staff, and Family Interviews

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Assessments of some of the symptoms and behaviour problems of long-term psychiatric patients living in the community were obtained independently from clinical interviews with such patients, and from interviews with day staff, residential staff and families caring for them. In general, interviews with residential staff and family members revealed much higher levels of symptoms and behaviour problems than either of the other two interviews. These findings have implications for research and clinical practice including the fact that adequate assessments should include the testimony of family or residential staff.

The increasing emphasis on care in multiple settings, whether in a combination of day and residential environments or because movement between settings occurs as needs change over time, has prompted questions as to whether methods of assessment that were satisfactory for long-term hospital care remain so in the new circumstances. Previously, ratings of negative symptoms based on a brief office interview were highly concordant with ratings of ward behaviour made by nurses following observation over a week (Wing, 1960, 1961*a*). Moreover, ratings of the ward behaviour made years apart remained highly correlated.

Such methods have been adapted for use in the community; for example, a series of social behaviour schedules (SBS) and social role performance schedules (SRPS) has been developed by the Medical Research Council (MRC) Social Psychiatry Unit for use in a variety of day and residential settings (Wing, 1989). There is evidence, however, that ratings can vary systematically according to the rater's expectations and experience (Wing, 1961*b*). If expectations also vary between respondents in different settings, they might differentially affect thresholds for reporting the evidence on which ratings are based. In addition, symptoms and behaviours may change as an individual moves from one setting to another, even on the same day. These changes may be spontaneous, or may reflect the fact that patients are under differing obligations and restrictions in different settings. Other factors that may influence rating are the amount of time raters have available to observe symptoms and behaviour, and the amount of disclosure about mental states that is made to or sought by raters.

Wing & Gould (1978) found that the parents of mentally handicapped and autistic children reported both more behavioural problems and more skills than did professional staff. The children spent more time in their own homes, received more individual attention there and tended to be more active than in the school, the nursery or any other day setting. Holmes *et al* (1982) confirmed the importance of setting even within a large mental hospital. Ratings made in the day training unit indicated fewer behaviour problems and higher skills than were recorded by ward nurses. The latter often did not know whether a resident could read or count or undertake various tasks. However, residents spent relatively little time in the training centres and nurses had more opportunity to observe disturbed behaviour in the less structured environment of the ward.

The effect of observation period is also evident in the lower reliability of ratings of behaviour based on a clinical interview using the Present State Examination (PSE) compared with those based on subjective experiences described by the person interviewed (Wing *et al*, 1974). The interview lasts about an hour and the examiner's rating of behaviour is based on this time only. The period described by the interviewee, however, is the past month.

There is a clear danger, therefore, that assessments made in out-patient clinics, day units or staff offices might not be a sufficient guide to the existence of real problems. This may particularly be true of differences between day and residential settings; the latter, of course, including the patient's own home. Several studies have recently documented the important role played by relatives caring for people with long-term psychiatric disabilities, and have noted their high level of objective burden and their low levels of contact with psychiatric services (Byrne *et al*, 1974; Creer & Wing, 1974; Creer *et al*, 1982; Gibbons *et al*, 1984; MacCarthy *et al*, 1989*b*).

This report is based on data from the Camberwell High Contact Survey (CHCS: Brewin *et al*, 1988; Brugha *et al*, 1988), which examined the needs of psychiatric patients in regular long-term contact with day-care facilities. For each patient in the study, different members of the research team conducted a clinical interview with the patient, an interview with day-care staff covering symptoms and behaviour during the previous month, and an interview with a relative or residential staff member where this was feasible. This design not only offered the opportunity of comparing the ratings made in office, day-care and residential settings, but estimates of symptoms and behaviour in a residential setting made by relatives could additionally be compared with estimates made by experienced residential staff.

Method

Full details of sampling and methodology are available in a previous report (Brugha *et al*, 1988). Briefly, the sample consisted of 145 patients aged 18 years or over who were ordinarily resident in South Southwark and who had been regularly attending a psychiatric day hospital or day centre for at least one year. In-patients, patients with severe mental retardation, patients addicted to substances other than alcohol, and patients over 65 years of age suffering from dementia were excluded. Approximately half the sample had received a hospital diagnosis of schizophrenia or paranoid psychosis. The median number of years since first contact with psychiatric services was 17 years for men and 14 years for women. The sample comprised 55% men and 45% women, and the average age was 50 years.

Each patient was interviewed using an early version of the 10th edition of the PSE by a research psychiatrist, who also administered other schedules for rating attitudes and social contacts. A detailed description of PSE10 is reported elsewhere (Wing *et al*, 1990). Interviews with day staff, and either with families or residential staff as appropriate, were conducted independently by a clinical psychologist or sociologist. Data were obtained from relatives for 53 patients and from residential staff for a further 25 patients. The interviews with family and staff members included the administration of a short version of the MRC Social Behaviour Schedule (SBS; Wykes & Sturt, 1986), a widely used measure of symptoms and behaviour problems. The particular items and scoring used are described elsewhere (Brewin & Wing, 1988).

The PSE10 and the SBS have a number of items with very similar content, although not all items are rated in identical ways in the two instruments. By collapsing across rating categories we derived four measures that could be scored in exactly the same way. These four measures were: (a) Retardation (two items: slowness, underactivity), maximum score = 4; (b) Neurotic symptoms (four items: depression, suicidal thoughts or acts, anxiety, obsessional behaviour), maximum score = 8; (c) Social interaction (three items: non-verbal behaviour, social mixing, initiation of conversation), maximum score = 6; (d) Behaviour problems (five items: overactivity, elation, odd gestures and mannerisms, acting out delusions and hallucinations, aggressive behaviour), maximum score = 10. These items represent only a small fraction of the total information recorded but they do allow limited comparisons to be made.

Interviewers underwent prior training on the SBS to ensure reliability of ratings between settings. As an additional check, 10 family interviews were tape-recorded and re-rated by another of the participating interviewers. Agreement between the interviewers on the above four measures was high (smallest $r = 0.97$). There was no evidence for any systematic differences in the levels of symptoms rated (largest $t = 1.5$, $P > 0.10$). Reliability on PSE10 scores was also high.

Results

As the data were all negatively skewed, non-parametric statistics are used throughout. First, scores obtained from family and residential staff members on the four measures were compared using a Mann–Whitney *U*-test to see whether there were any between-group differences in the reporting of symptoms and behaviour problems. There were no significant differences in the reporting of retardation ($\bar{X} = 1.08$ v. 1.26), social interaction difficulties ($\bar{X} = 2.62$ v. 2.10), or behaviour problems ($\bar{X} = 1.52$ v. 1.59). However, family members reported more neurotic problems than did residential staff ($\bar{X} = 1.14$ v. 0.62, $z = 2.11$, $P < 0.05$). In view of the predominantly non-significant differences, data supplied by family and residential staff members are treated as belonging to a single group in most of the following analyses.

TABLE I

Mean ratings of symptoms and behaviour problems by clinical interview, interview with day staff, and interview with family/residential staff

Symptom/behaviour problem	*Mean rating*[1]			χ^2	P<
	Clinician	*Day staff*	*Residential staff/family*		
Retardation	0.15a	0.43b	1.13c	14.59	0.001
Social interaction	0.78a	1.31b	2.46c	11.31	0.01
Neurotic problems	0.75a	0.48b	0.99a	6.12	0.05
Behavioural problems	0.73a	0.65a	1.54b	11.35	0.01

1. Different letters (a, b, c) in the rows indicate significantly different scores ($P < 0.01$) (e.g. the three ratings of retardation were all significantly different while the clinicians' ratings of neurotic problems were only significantly different to those of the day staff).

TABLE II

Agreement between clinical interviews, interviews with day staff, and interviews with family/residential staff (table shows percentage agreement and weighted kappa averaged across individual items)

	Clinician/ day staff (n=*114*) %	k	*Clinician/ residential* (n=*43*) %	k	*Day staff/ residential* (n=*43*) %	k
Retardation (2 items)	84	0.18	63	0.07	59	0.16
Social interaction (3 items)	65	0.15	45	0.00	51	0.16
Neurotic problems (4 items)	85	0.25	79	0.13	79	0.16
Behavioural problems (5 items)	84	0.30	78	0.21	77	0.21

We next carried out within-subject analyses to compare scores on the four measures obtained from the clinical interview, the interview with day staff, and the interview with family/residential staff. Mean scores from these three interviews are shown in Table I.

Overall differences between the three sets of interview data were tested using four Friedman non-parametric analyses of variance, which yielded the χ^2 values shown in the penultimate column of Table I. The difference between individual pairs of scores was investigated with Wilcoxon tests. Table I indicates that interviews with family/residential staff produced significantly more reports of slowness and underactivity, impaired social interaction, and behavioural problems than did either of the other interviews. Family/residential staff interviews also provided more evidence of neurotic problems than did interviews with day staff. Separate analyses of variance comparing scores on neurotic symptoms from the clinical interview, day staff and residential staff, and from the clinical interview, day staff and family member, indicated that this effect was a function of family interviews ($\chi^2=6.19$, $P<0.05$), not of residential staff interviews ($\chi^2=1.44$, $P>0.10$).

In addition, Table I shows that interviews with day staff yielded significantly more evidence of slowness and underactivity, and impaired social interaction, than did the clinical interview. In contrast, clinical interviews led to the identification of significantly more neurotic symptoms than did interviews with day-care staff.

Level of agreement between the three sets of interview data was next investigated using both a percentage agreement and the weighted kappa statistic, which corrects for the amount of agreement to be expected by chance. Table I shows these data, averaged across individual items, for each of the four subscales. Bearing in mind that the distribution on all items was quite heavily skewed, levels of agreement were poor. Agreement on the items measuring neurotic and behavioural problems was generally greater than agreement on those measuring retardation and social interaction. As would be expected from our other results, agreement between the clinical interview and the day-staff interview was almost invariably higher than between the residential interview and either of the other two interviews.

Discussion

The results show that estimates of some symptoms and behavioural problems are highest when they are made by those who can observe the patient in a residential setting. This might be because raters in residential settings have higher expectations of patients and over-report symptoms and behaviour problems. However, this seems unlikely since the same patterns of observations are made by experienced residential staff and by family members. Rather, it is probable that the discrepancy between day and residential settings is due either to greater opportunities for observation, or to genuine differences in the behaviour shown in the two settings, or to both. The implication is that adequate assessments must include the testimony of family or residential staff if the degree of these problem behaviours is not to be seriously underestimated.

Whereas interviews with day staff invariably produced lower estimates of problem behaviours than did residential interviews, clinical interviews gave lower estimates of retardation and social interaction deficits than did either of the other assessments, probably owing to the reduced opportunities for observation. Estimates of behaviour problems were also lower in clinical interviews than in residential interviews, and thus it is suggested that PSE10 should be supplemented by a schedule such as the brief SBS when rating patients with long-term disablement. The estimate of neurotic problems given by the clinical interview, however, was equivalent to that provided by the residential respondents, and significantly greater than that provided in the day setting. We may speculate that patients disclosed their mental states both to their families and in response to the clinical interview, but were not routinely called upon to make such disclosures by day staff.

A related cause for concern was the low agreement between the different assessments, which is not altogether surprising given the discrepancies in the levels of symptom reporting. Reliability between day and residential settings was substantially poorer than equivalent figures given for a different version of the SBS by Wykes & Sturt (1986), but this may be because our assessments were conducted by independent interviewers whereas Wykes & Sturt employed the same interviewer in both settings. A partial solution to the problem of low reliability may be to supplement staff interviews with more extensive questioning of patients themselves, since there is evidence that patients with long-term psychiatric disabilities can be reliable informants on some topics if questioned appropriately (MacCarthy *et al*, 1986). Another implication of our findings is that there should be more intensive interaction between day staff and patients' families and hostel staff. A suitable forum for relatives may be a counselling group, and one such group has been shown to be both low in cost and clinically effective (Kuipers *et al*, 1989; MacCarthy *et al*, 1989*a*).

We believe our findings have considerable significance both for clinicians and researchers. Community care workers who look after chronically disabled patients need to know the limits of the assessments made during restricted periods of observation in an office or day centre. Researchers investigating the impact of different treatments, or of different forms of community care, also need to consider how they should measure clinical outcomes if they are to obtain more complete estimates of symptoms and problem behaviours.

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The Attitude of Parents to the Use of Computer-Assisted Interviewing in a Child Psychiatry Service

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Before undergoing interview, parents reported that a computer-assisted interview would lack the friendly and personal approach of one conducted by a clinician but, after completing the interview, parents showed more acceptance of the procedure. Computer-assisted interviewing has the potential to play an important role in the collection of clinically relevant information from both parents and children referred to child psychiatry services.

Computer-assisted interviewing has been used in a range of mental health settings with adult patients (Slack & Van Cura, 1968; Lucas *et al*, 1977; Carr *et al*, 1981; Carr & Ghosh, 1983); however, little use has been made of the procedure in child psychiatry services, where it could enable children or parents to enter information directly into a computer terminal. Employed in this way, computer-assisted interviewing has the potential to provide a reliable and economical method of collecting information about the nature and severity of disorders in referred children. Information collected could be made available to clinicians immediately in the form of computer-generated summary reports describing individual patient problems. In addition, it could be stored and later used to describe the overall pattern of childhood disorders referred to a service.

In Australia, there is a shortage of the trained clinicians needed to assess the large numbers of children referred to child psychiatry services (Royal Australian and New Zealand College of Psychiatrists, 1982; Smith, 1983). Computer-assisted interviews could help alleviate this problem by both establishing the urgency of need at the time of initial referral and aiding clinicians in later assessment. The latter is important because it provides clinicians with direct support, and may help overcome past resistance to the introduction of computer technology into clinical services. Employed properly, computer-assisted interviews cause minimal disruption to clinical services. In addition, the use of printed summary reports overcomes problems of poor legibility, and the time and costs of coding and scoring written questionnaires (Klein *et al*, 1975; Byrnes & Johnson, 1981).

A prerequisite to the successful use of computer-assisted interviews in child psychiatry services is parent satisfaction with the procedure. Despite its importance, however, a literature search revealed only one study that had evaluated parent satisfaction with a computer-assisted interview used in a child psychiatry service (Coddington & King, 1972). Mothers of children were interviewed directly using a computer terminal; acceptance of the procedure by mothers was encouraging and most had no difficulty in coping with the interview.

The aim of this study was to investigate parent satisfaction with a computer-assisted interview employed to assess children referred to a child psychiatry service. The study compared satisfaction with the computer-assisted interview and satisfaction with two other methods of collecting information, clinical interview and written questionnaire.

Method

Informed consent to participate in the study was sought from all parents of children referred to the Department of Psychiatry at the Adelaide Children's Hospital over six months. Children are referred to the department by a wide range of community and hospital services. All referred children are seen by a psychiatrist or psychologist and the nature of their clinical assessment varies depending on the nature of their problem and the philosophy and training of their clinician.

Two parents who were illiterate and three who did not speak English were not included in the study, leaving 161 parents eligible to participate, of whom 140 (87%) agreed to participate. Subsequently, 29 of these parents were not able to undertake their interview because they lacked the necessary time on the day of their assessment.

When parents and children arrived in the Department of Psychiatry they were met by a research assistant who had earlier contacted them by telephone or letter to advise them of the study. A random-number chart was used to assign parents to one of two groups: computer-assisted interview, or written questionnaires. All data collection took place *after* the parents and their children had been seen by clinicians and, except for minor differences required by the two collection methods, the content of the interviews was identical.

In the computer-assisted interview, Hewlett Packard 150 terminals fitted with touch-sensitive screens enabled interviewees to record their response by simply touching the screen at the appropriate position. Two terminals were used in the study and both were located in a room adjacent to the Department of Psychiatry. (To ensure privacy, the room was divided.) The research assistant was available to answer questions but after orientating parents his help was rarely needed.

Upon completion of the interview, data from the computer-assisted interview and written questionnaires were immediately scored and a printed summary report made available to the clinician who had assessed the child.

Instruments

For the purpose of the computer-assisted interview, three questionnaires, measuring emotional and behavioural problems in children, family adjustment and recent stressful events, were converted into a format suitable for use with a computer terminal. Written questionnaires were bound together to form a single questionnaire which could be readily managed by parents.

Emotional/behavioural problems were identified using the behaviour problem section of the Child Behavior Checklist (Achenbach & Edelbrock, 1983), which consists of 118 behaviour problem items answered using a three-point response scale labelled "very true or often true", "somewhat or sometimes true", and "not true". The checklist provides an overall rating of behaviour problems and separate ratings in two broad areas that Achenbach & Edelbrock (1983) have labelled "internalising" and "externalising". Ratings of behaviour in specific areas can also be obtained. The Child Behavior Checklist has been widely used with both clinic and community populations, and extensive information is available with regard to its reliability and validity (Achenbach & Edelbrock, 1983).

Family adjustment was assessed by means of the general functioning scale of the Family Assessment Device (Epstein *et al*, 1983). The Family Assessment Device is a self-report measure of family functioning which is composed of six subscales measuring specific areas of family functioning and a 12-item general functioning scale which provides an overall measure of the health/pathology of the family. The general functioning scale has been shown to have good reliability and validity (Miller *et al*, 1985; Byles *et al*, 1988).

A questionnaire used to identify recent stressful events to which the referred child may have been exposed was developed at the request of clinicians participating in the study. Clinicians felt that this was important information which could be readily obtained in a screening interview. The questionnaire was derived from existing measures of stressful events (Coddington, 1972*a*,*b*) and consists of 12 common stressors answered yes or no. Finally, at the end of the assessment, all parents were given the opportunity to provide written information describing their child's problems. Parents completing the computer-assisted interview did this by typing in comments while parents completing the questionnaire wrote in their comments.

Parent satisfaction was evaluated by means of two approaches. Firstly, parents ranked from one to four their preference for each of four alternative assessment methods: assessment with a clinician, assessment using a written questionnaire, assessment using a computer terminal, and assessment by both a clinician and computer terminal. This was done by parents both before and after the clinician and other assessment. Secondly, a semantic differential scale (Osgood, 1957) employing 12 adjective pairs rated on a five-point scale was used to evaluate parents' perceptions of their interviews. Items for the scale were selected from scales previously used to measure satisfaction with computer-assisted interviewing in adult services (Lucas *et al*, 1977; Skinner *et al*, 1985). This scale was completed by parents immediately after their clinical interview, and again immediately after completing the computer-assisted interview or written questionnaire. In order to avoid any possible method variance, information about parent satisfaction was all collected by means of written questionnaires.

Results

There were no significant differences between the demographic characteristics of the two groups of parents (Table I).

The preference for four alternative assessment methods reported by parents are shown in Table II. The ranking given to each assessment method was collapsed into one of two categories consisting of first and second preferences, and third and fourth preferences. McNemar's statistic (Armitage & Berry, 1987) was used to test the significance of changes in preferences after parents had completed their particular assessment procedure. The small loss of subjects in the table was a result of some items on the questionnaire not being completed by parents. A significant number of parents who completed the computer-assisted interview reported an increased preference for "assessment with both a clinician and computer terminal" and "assessment using a computer terminal", and a decreased preference for "assessment using a written questionnaire". In contrast, there were no significant changes in the preferences reported by parents who completed the written questionnaires.

TABLE I
Demographic characteristics of parents

	Computer-assisted interview group (n=56)	*Written questionnaire group (n=55)*
Sex distribution		
male	3	1
female	53	54
Age: years		
mean	34.0	33.6
s.d.	5.9	7.7
Clinician seen		
psychiatrist	25	20
psychologist	31	35
Occupational class[1]		
1–3	6	3
4–6	8	3
7–8	25	20
missing	17	29

1. Daniel (1983).

TABLE II
Preferences assigned by parents who completed the computer-assisted interview

Assessment method	*Pre-assessment preference*		*Post-assessment preference*	
	1–2	*3–4*	*1–2*	*3–4*
Parents who completed the computer-assisted interview (n = 53)				
Computer terminal**	5	48	16	37
Clinician and computer terminal*	30	23	37	16
Clinician	48	5	44	9
Written questionnaire**	23	30	9	44
Parents who completed the written questionnaire (n = 51)				
Computer terminal	5	46	7	44
Clinician and computer terminal	28	23	26	25
Clinician	45	6	44	7
Written questionnaire	24	27	25	26

*$P<0.05$, **$P<0.01$.

The Kolmogorov–Smirnov two-sample test (Siegel, 1956) was used to test for the significance of differences between scores on the semantic differential scales from both groups combined (Fig. 1). This non-parametric test was chosen because the response categories are clearly discrete rather than continuous and the test makes no assumptions about the distribution of responses. There were significant differences ($P<0.05$) between the scores describing the computer-assisted interview and the clinical assessment on five of the dimensions on the semantic differential scale: human-like–mechanistic, friendly–cold, personal–impersonal, pleasant–unpleasant, and accurate–inaccurate. In each case, the clinical interview was rated more favourably. There were also significant differences between the scores describing the written questionnaires and the clinical assessment on the initial four of these dimensions. Once again, in each case the clinical assessment was rated more favourably. Finally, in the comparison of scores describing the computer-assisted interview and the written questionnaires, the computer-assisted interview scored significantly less favourably on the dimensions human-like–mechanistic and personal–impersonal.

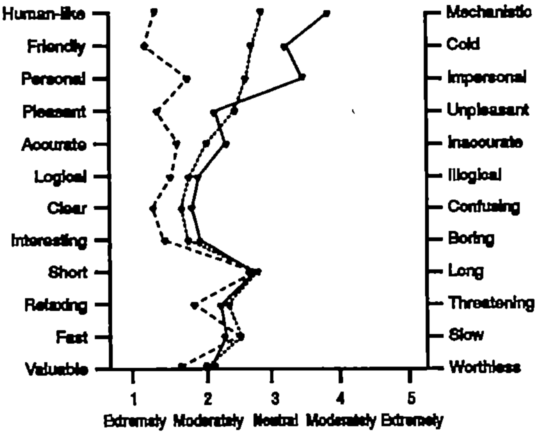

FIG. 1 Mean scores for three assessment methods on the semantic differential scale for parents (●——●, computer-assisted interview; ●·····●, written questionnaire; ●—●, clinical interview).

The time taken to complete and score the computer-assisted interview was compared with that for the written questionnaires using paired *t*-tests to determine the significance of differences. Although the computer-assisted interview took signficantly longer to complete than the written questionnaire (26.09 v. 19.23 minutes, $t=4.04$, d.f. = 102, $P<0.01$), when the time taken to score the two procedures was taken into account (3.64 v. 16.65 minutes for the written questionnaire) the total time required for the hand-scored written questionnaire was significantly longer than that of the computer-assisted interview (35.91 v. 29.73 minutes, $t=3.20$, d.f. = 102, $P<0.01$). There was no signficant difference ($t=0.67$, d.f. = 102, $P=0.522$) in the total time required when the written questionnaire was scored using the computer scoring program (29.73 v. 30.87 for the written questionnaire).

Finally, the number of written/typed comments provided by the parents completing the two procedures was compared. This comparison showed that 82% of parents who completed the written questionnaires provided written comments compared with only 30% of parents who completed the computer-assisted interview.

Discussion

Many child psychiatry services routinely collect demographic and clinical information from parents at the time of a child's referral. Such information is used to provide support for clinicians and for administrative and planning purposes. Unfortunately, problems of legibility, lack of the staff time required for storage and retrieval of information, and the complexities of questionnaire scoring often result in the information being underused.

Computer-assisted interviewing offers an efficient method by which information can be routinely collected from parents and subsequently made available to clinicians or administrators in the form of printed reports. After it is collected, clinical information can be immediately scored by computer and made available to clinicians to aid in the assessment of individual children and their families. The information can also be stored and later retrieved in the form of summary reports showing the overall pattern of childhood and family problems referred to a service.

It is important to realise that the collection of information by means of a computer-assisted interview need not be restricted to information from parents. Children whose parents participated in this study were themselves keen to participate in the computer-assisted interview. This is an important observation because there are significant differences in reports about childhood emotional and behavioural problems provided by children and their parents (Achenbach *et al*, 1987). Computer-assisted interviewing could readily be adapted to collect information simultaneously and independently from children and their parents, and areas of agreement and disagreement could then be identified.

Despite the potential advantages offered to child psychiatry services by computer-assisted interviewing, it is important to note that results from this study indicate that parents prefer an interview with a clinician. Results obtained from the semantic differential scale reveal that parents perceive a clinical interview to be more friendly and personal than a computer-assisted interview. These findings are very similar to those of Skinner *et al* (1985) whose computer-assisted interview was specifically designed to be friendly, giving participants significantly more feedback during the course of the interview. The consistency of results in the two studies suggests that it may be difficult for computer-assisted interviews to replicate the personal and friendly qualities of a clinical interview. Instead, it may be more productive if future studies concentrate on trying to develop the potential of computer interviews to support the information requirements of clinicians and administrators through their ability to systematically collect, store, analyse and retrieve demographic and clinical information (Sarris & Sawyer, 1990).

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The Interval General Health Questionnaire

P. G. SURTEES and P. McC. MILLER

An adaptation of the LIFE procedure was used with a self-report questionnaire to assess 173 medical students, close to their enrolment and again about six months later. The assessment procedure enabled changes in symptoms of anxiety and depression over the interval between interviews to be recorded and the subsequent classification of the symptom patterns into course categories. While women scored markedly higher than men at initial assessment, this was not so at follow-up. Almost 25% of students reported the co-occurrence of at least five psychological symptoms at some time during the six months. For many students these were of a persistent nature. The adapted GHQ meets the dual demands of restricted interview time and the need to assess the more minor psychological conditions.

One of the enduring problems confronting psychiatry concerns how best to measure the very phenomena upon which the discipline is based, and in recent years this measurement problem has become a dominant feature of the research literature. Central to this effort is the fundamental and reasonable concern with applying universal standards to provide a rational framework for the continued evolution of the assessment and classification of psychiatric phenomena. Of course it will always be the case that particular assessment techniques may be best suited to particular settings and that developments in psychiatric nosology may have only tangential or, at best, uncertain relevance to large bodies of activity in psychological research. An alternative but equally legitimate strategy of inquiry to that involving the use of diagnostic concepts, has relied upon those assessment methods whose primary objective is to provide a simple enumeration of symptoms at a particular time or during a particular period. Such methods usually are based on the self-report of symptoms by questionnaire and thereby carry with them certain hazards concerning their meaning and interpretation. Nevertheless, they are widely used and have been found to be of particular value, especially for screening purposes, in identifying individuals with an increased likelihood of a current psychiatric disorder.

Both standardised psychiatric interviews and self-report assessments, in different ways, offer complementary approaches which are broadly adequate to meet the range of measurement requirements concerning 'current' psychiatric status. A continuing challenge is to provide viable solutions to the problem of assessing *change* in psychiatric status over time, be it with reference to a diagnostic scheme or for more minor, perhaps rather transient changes in symptom state. The development of standardised psychiatric interviews reflects the continuing evolution of psychiatric diagnostic schemes, with new criteria being embodied in the interviews as they are agreed upon. In general, while there has been a preoccupation with the development and imposition of standardised diagnostic criteria, there has been a relative neglect of measures specifically designed to document the *course* of episodes of psychiatric disorder. One outstanding exception has been the work originally initiated by Dr Robert Shapiro and continued by Dr Martin Keller. The interviewing technique they developed, the Longitudinal Interval Follow-up Evaluation (LIFE), was designed for a multicentre study of depressive illness (Keller *et al*, 1987). A special feature of this approach is that it assesses variation over time of the 'psychiatric status' of the interviewee in accord with the particular diagnostic criteria and scheme chosen for study. The method has facilitated a detailed examination of, for instance, factors associated with the recovery and relapse of major depressive disorder (e.g. Keller *et al*, 1982*a*,*b*) and with the risk of chronicity (Keller *et al*, 1986).

The present paper reports on the application of some of the principles of the LIFE method to the assessment of the course and change in minor spells of psychiatric distress and thereby seeks to help bridge the divide between advances made in formal psychiatric interview assessments and self-report methods. Through this combination of methods, it is also hoped to underpin the advantages already demonstrated for the criterion as opposed to the conventional aggregate scoring procedure of a symptom check-list (e.g. see Uhlenhuth *et al*, 1983; Murphy *et al*, 1985, 1987; Murphy, 1986).

As part of a study designed to investigate the interrelationship between health, coping resources, social support, social adversity and personality in newly enrolled medical students, an opportunity arose to evaluate a method of documenting change over time in self-reported symptoms. This part of the study had the following specific aims:

(a) to assess the prevalence of psychiatric symptoms in newly enrolled medical students at entry and over the ensuing six months of their training, with a special emphasis being given to the examination of sex differences
(b) to classify students into categories on the basis of the patterns of reported symptoms over time and to investigate whether there is a sex-specific differential adaptation to entry to medical school
(c) to evaluate an aggregate versus a criterion-based approach to summarising change in psychiatric symptoms over time
(d) to determine the six-month inception rate of spells of psychiatric symptoms and to identify whether there are sex differences in these rates.

Method

Following pilot work and interviewer training, all first-year medical students at the University of Edinburgh were approached for interview within four weeks of their enrolment. The interview included sections dealing with the following areas: demographic factors, health, social contacts, coping resources, and personality. All students seen at the initial interview were approached approximately six months later for a follow-up interview. This paper is concerned with only one component of the assessment, psychological state.

As part of the assessment, the 30-item General Health Questionnaire (GHQ; Goldberg, 1978; Goldberg & Williams, 1988) was administered, both at the initial and follow-up interviews. Since its development, the GHQ has been used in a variety of forms (e.g. from 12–60 items) to facilitate the detection of psychiatric disorder in a wide variety of settings and cultures (e.g. Tarnopolsky *et al*, 1979; Banks, 1983; Chan & Chan, 1983; Mari & Williams, 1985; Bridges & Goldberg, 1986; Williams *et al*, 1986; Boardman, 1987). Its choice for this study ensured comparison in some respects with other work, but the strategy to be described below in relation to particular symptoms is generally applicable and therefore need not be tied or limited to the GHQ.

The principles of the LIFE method depend in part upon determining the times of change in psychiatric status ratings (PSRs) within a study interval. The PSRs relate directly to the extent to which diagnostically significant symptoms are present and co-occur. Ratings made at one interview are 'drawn forward' at follow-up, based upon new information gathered at the follow-up interview. The interviewer seeks to identify only those times which correspond to *changes* in PSRs. For present purposes, this principle was adopted though limited to certain symptoms selected from the GHQ. Our primary objective therefore was to be able to document the changes in those symptoms over the study period.

The process of selecting items from the GHQ-30 was guided not only by the results of published papers but also by our desire to impose a criterion-based scoring method. Two subsets of six items each were selected from the 30-item GHQ that were considered to be broadly related to depressive and anxiety conditions. Guidance in selecting items was obtained from the manual of the GHQ (in particular results concerning the factor structure of the questionnaire) and from those symptoms that are commonly included within diagnostic criteria for depressive and anxiety disorders and for which the GHQ-30 contained appropriate questions. Several papers provided results which influenced item choice.

Goldberg & Williams (1988) have reported the results of a number of principal-component analyses of the GHQ-30 and GHQ-12. Between 48% and 71% of the total variance was accounted for, in the case of the GHQ-30, by from four to ten significant components. Of these studies, one undertaken by Goldberg *et al* (1976) on 1310 United States general practice patients accounted for 51.5% of the total variance using the 30-item GHQ. The first of the four significant factors in the varimax rotation accounted for 20.6% of the variance and was labelled 'depression and anxiety'. Subsequently, Goldberg & Hillier (1979) published a scaled version of the GHQ based upon a varimax rotation of the 60-item GHQ, 48% of the variance being accounted for by four scales each consisting of seven questions; of these scales, scale B was labelled 'anxiety and insomnia', and scale D 'severe depression'. The six chosen 'anxiety' items were in close accord with those items comprising scale B in the 28-item GHQ (scale B item 2 being taken to be approximately equivalent to item 3 on the GHQ-30).

Only four of the items chosen to represent our 'depression set' formed part of scale D of the 28-item GHQ (items 24, 25, 29 and 30). For reasons similar to those guiding choice of the anxiety-related items, two additional depression-related items were also included (item 22 "Been feeling unhappy and depressed?" and item 23 "Been losing confidence in yourself?" – items G1, sensitivity 86%, and G2, sensitivity 78%, respectively in the original factor analyses).

In addition to the above findings, the results obtained from the latent-trait analysis of symptom data (whether obtained from formal psychiatric assessments (see Goldberg *et al*, 1987, Table 4) or based on the GHQ (see Duncan-Jones *et al*, 1986, Table 2) provided additional support for item choice. It should also be noted that the overlap between our final choice of items and the 12-item GHQ was limited to only five questions.

The two sets of selected items were cast into a computer-generated pro-forma that included the GHQ item number, the wording of each chosen item, the individual student scores for the items at initial interview, the date of initial interview and columns to allow the follow-up scores to be recorded (see Appendix). Upon completion of the follow-up GHQ, the interviewer was required to record the scores of each of the selected items on the personalised pro-forma and then follow the interview procedure for completion of the ratings covering the follow-up interval. Symptom levels at the two interviews provided anchor points to facilitate establishing each student's symptom status over the study interval. Students were not directly questioned about each

TABLE I

GHQ and I-GHQ scores at initial and follow-up interviews, by sex

	Initial interview			*Follow-up interview*		
	All (n = *175)*	*Males only* (n = *96)*	*Females only* (n = *79)*	*All* (n = *173)*	*Males only* (n = *95)*	*Females only* (n = *78)*
Scale scores: mean (s.d.)						
GHQ	5.93 (5.85)	4.58 (4.69)	7.56* (6.69)	4.70 (5.89)	4.56 (5.41)	4.87 (6.45)
I-GHQ:						
all items	2.44 (2.49)	1.80 (1.93)	3.22* (2.85)	1.76 (2.43)	1.72 (2.18)	1.81 (2.72)
anxiety set	1.69 (1.49)	1.38 (1.36)	2.08* (1.57)	1.19 (1.56)	1.21 (1.47)	1.17 (1.67)
depression set	0.75 (1.22)	0.43 (0.79)	1.14* (1.51)	0.57 (1.05)	0.51 (0.93)	0.64 (1.18)
Percentage scoring at or above cut-off point						
GHQ: % scoring ≥5	49.7	40.6	60.8	35.8	34.7	37.2
I-GHQ						
all items, % scoring ≥2	53.7	41.7	68.4	37.6	40.0	34.6
anxiety set, % scoring ≥2	47.4	36.5	60.8	32.4	35.8	28.2
depression set, % scoring >0	38.3	28.1	50.6	32.9	31.6	34.6
'Sensitivity'[1]	90.8	87.2	93.8	88.7	87.9	89.7
'Specificity'[1]	83.0	89.5	71.0	91.0	85.5	98.0

1. Relationship between I-GHQ (all items, cut-off point ≥2) and GHQ (usual cut-off point of ≥5) as criterion.
*$P<0.001$.

month of the study period in order to establish symptom ratings – questions were directed only towards identifying those times when changes in the selected symptoms had occurred which corresponded to the GHQ scale points.

Two scoring methods were adopted for this study. In the first, symptoms were scored according to the conventional binary scoring (0 or 1) of GHQ items; in the second, a criterion-based approach was adopted. In order to apply the second method, two items were endowed with greater significance than others to reflect the importance usually placed upon them in diagnostic schemes. The item "Been feeling nervous and strung-up all the time" (item 28 GHQ-30; item B7 scaled GHQ) was chosen as the 'pivotal' item for the 'anxiety set' because it appeared to be a reasonable proxy for the key questions used to establish generalised anxiety disorder by formal psychiatric interviews imposing the Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978) or DSM-III-R criteria (American Psychiatric Association, 1987). This particular item (under conventional GHQ scoring) appears to have acted as an effective discriminator of the 'severely ill group' in the original GHQ item analysis (see Goldberg & Williams, 1988; Appendix 1, item G11, item sensitivity 72%). Similar reasons influenced the choice of item 22, "Been feeling unhappy and depressed?", selected as the key criterion item for the set of depression-related symptoms.

An example of a completed form, together with the associated interviewer guide notes, is given in the Appendix. This interval form of the questionnaire will be referred to as the 'I-GHQ'.

Results

Of the 181 students eligible for inclusion, interviews were successfully obtained from 175 (96.7%) within four weeks of their enrolment. Five to six months following the initial interview all students were approached once again for a follow-up assessment, and 173 interviews (98.9%) were completed. All of the students were single and they ranged in age from 17 to 25 years with only three aged over 20 years.

The results shown in Table I were obtained using the conventional binary scoring (0 or 1) of all the 30 GHQ items and a similar method of scoring the 12 items of the I-GHQ. While at initial interview the female students reported symptom levels significantly higher ($P<0.001$) than the males on all measures shown in Table I, the difference was no longer present at follow-up. Further indications of the extent to which this student sample scored highly on the full questionnaire are given by Fig. 1. The pattern of results revealed by the GHQ was repeated for the I-GHQ.

Also in Table I, the conventional cut-off point applied to the 30-item GHQ is contrasted with others based on the I-GHQ. At initial interview almost half the students scored at or above the conventional cut-off points, while at follow-up about 35% were still at or above this level. Estimates of 'sensitivity' (in this instance the proportion of 'GHQ-30 cases' correctly identified) and 'specificity' (the proportion of 'GHQ-30 normals' correctly identified) associated with the chosen cut-off point on the I-GHQ are also shown in Table I to indicate the extent to which the same students are being identified by both forms of the questionnaire.

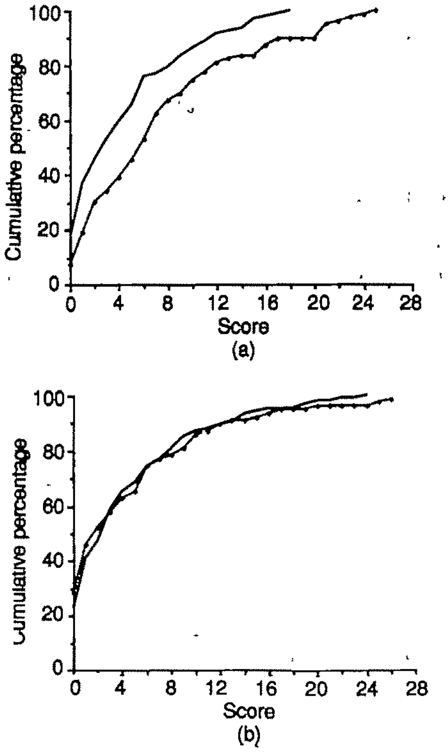

FIG. 1 Cumulative distribution by sex of scores on the 30-item GHQ at both (a) initial and (b) follow-up interviews (——males, $n = 96$; ——females, $n = 79$). A similar pattern was shown with the 12-item I-GHQ.

The results show considerable similarity – perhaps surprising given the brevity of the I-GHQ.

Students were also classified according to the criterion-based approach. In view of the empirical nature of the exercise, several grades of criterion severity were established. The initial (general) criterion adopted was the presence of either (or both) of the two 'pivotal' symptoms, "Nervous and strung-up all the time" and "Feeling unhappy and depressed", with or without any additional symptoms of the ten remaining (all rated according to the conventional GHQ binary scoring): this was considered sufficient within any of the interval monthly time frames to signal a "spell of psychological symptoms" at that time. Subsequently, three criterion levels were imposed where specific numbers of additional symptoms (similarly scored) had to be present in addition to depression and/or anxiety. The first of these involved none or one additional symptom (out of the 10 possible), the second two or three and the third four or more additional symptoms.

The first analysis therefore focused upon the initial (general) criterion mentioned above and (with adjustment when necessary for variation in individual student follow-up times) classified the pattern of ratings according to the categories shown in Table II. For example, a student who at initial interview did not satisfy the minimum criterion but at follow-up reported the necessary symptoms to be present and to have commenced within the study period, would be allocated to the 'new and on-going' subgroup. The category 'multiple' refers to those students who reported several discrete spells of symptoms with each spell meeting the necessary general criterion and being separated by times when the pivotal symptoms were not present. While there was an overall sex difference in the distribution of patterns ($\chi^2 = 12.48$, d.f. $= 5$, $P = 0.029$), it is clear that those patterns potentially of greatest concern were of broadly equal prevalence among the sexes. Similar percentages of male and female students were found to have experienced anxiety and/or depression continuously for most of the follow-up (20.0% of the males and 23.1% of the females).

The degree to which the students fulfilled the more demanding symptom criteria is shown in Table III, which shows that a considerable proportion of this new intake of medical students reported experiencing multiple psychological symptoms at the same time, and that for a sizeable subgroup they were of an enduring nature. Almost a quarter of all students (and a third of female students) had spells of symptoms that included depression and/or anxiety together with four or more additional symptoms, and that lasted for 9.3% of the study time (representing about eight student years). The female students experienced spells of symptoms that were particularly long lasting.

TABLE II

'Spell' course status as determined by the general criterion[1], by sex

	All		*Males only*		*Females only*		*Male:female*
'Spell' course status	n	%	n	%	n	%	*% ratio*
Well	65	37.6	46	48.4	19	24.4	1.98:1
Recovering	30	17.3	11	11.6	19	24.4	0.48:1
New and recovery	21	12.1	9	9.5	12	15.4	0.62:1
New and on-going	20	11.6	10	10.5	10	12.8	0.82:1
Multiple	22	12.7	12	12.6	10	12.8	0.98:1
Continuous	15	8.7	7	7.4	8	10.3	0.72:1
Totals	173	100	95	100	78	100	

[1] Report of either of the two pivotal symptoms, "Nervous and strung-up all the time" or "Feeling unhappy and depressed".

TABLE III
Fulfilment of various 'spell' criterion levels over the entire study time, and six-month inception rates

Sample	*Depression and/or anxiety present together with a number of additional symptoms*			
	0 or 1	*2 or 3*	*4 or more*	*Totals*
Overall 'morbidity' levels: %				
All ($n=173$)				
% meeting each criterion at least once	24.9	38.7	24.9	62.4
mean % of entire study time at each criterion	7.6	13.1	9.3	30.0
Males only ($n=95$)				
% meeting each criterion at least once	19.0	33.7	17.9	51.6
mean % of entire study time at each criterion	5.0	13.4	5.8	24.2
Females only ($n=78$)				
% meeting each criterion at least once	32.1	44.9	33.3	75.6
mean % of entire study time at each criterion	10.8	12.7	13.4	36.9
Six-month inception rates: %				
All ($n=113$)	19.5	23.9	10.6	42.5
Males only ($n=70$)	14.3	18.6	8.6	34.3
Females only ($n=43$)	27.9	32.6	14.0	55.8

Percentages are not additive across criterion levels.

As a further indicator of the degree to which these students experienced psychological symptoms, analyses were undertaken to determine the six-month inception rates for each of the criterion-based levels of symptoms. Inception for this study refers to those students who developed spells of symptoms (at each criteria level) for the *first time* during the period of observation. The inception rates therefore exclude those students who at initial interview fulfilled any of the criterion levels being tested. The results are also shown in Table III, for the overall group and separately by sex. It shows for example that, of those 43 female students who were not bothered by either depression and/or anxiety at the time of the initial interview, 14.0% went on to experience symptom spells that not only included depression or anxiety, but four or more symptoms in addition. The inception rate of symptom spells fulfilling this criterion for all students was 10.6 per six months per 100 students.

Discussion

Inevitably many issues remain to be addressed. These concern the selection of items from the GHQ-30 and how they were subsequently analysed, the relationship between the I-GHQ, the GHQ-30 and clinician-based measures of disorder. Questions arise from the substantive findings, in particular how they compare with other populations of a similar age and what may be the advantages of using the I-GHQ beyond one of the more usual forms of the GHQ.

The results of this study have shown that the I-GHQ appears to perform in much the same manner as its 'longer parent' in conventional cross-sectional analyses. A further and much more detailed discussion of the relative performances of the two scales in relation to external clinical psychiatric status forms the basis of the following paper (this issue, pp. 686–693).

This study has clearly indicated the extent to which newly enrolled medical students experience spells of distress during their first few months at medical school. As the students provided the first opportunity to evaluate the I-GHQ, it has not been possible to compare the profile of 'course' categories obtained in this study with any others. However, the GHQ scores at each interview provide one means of comparison. Among 791 British 18–24-year-olds included in a random sample of over 6000 respondents (Cox *et al*, 1987) who completed the GHQ-30, female scores exceeded those of males (females: mean score 4.86, 38% scored ≥5; males: mean score 3.53, 30% scored ≥5). Comparison of these figures with those shown in Table I reveal that while the scores of the medical students at enrolment were markedly higher than the broadly age-equivalent random sample, at follow-up only the male students still remained relatively high scoring.

A further comparison is with a study by Firth (1986) in which 318 medical students in their fourth year at the universities of Sheffield, Manchester and Leeds completed the 12-item version of the GHQ and were compared with a sample of 222 young employed people. The estimated prevalence of psychological disturbance among the students was 31.2% compared with 9.7% in the young employed. A follow-up of a sample of 170 of the same medical students during their pre-registration year revealed that 50% then had scored four or above on the GHQ-30 (Firth-Cozens, 1987). A study of 523 medical students from the University of Belgrade (Eric *et al*, 1988) assessed both close to enrolment and two years later using the GHQ-60, and supplemented by a psychiatric interview, revealed an overall point-prevalence rate of psychiatric disorder at enrolment of 16.1% (females 21.6%) and two years later 17.5% (females 22.7%).

Evidence of the extent to which medical students in the initial stages of their course meet psychiatric diagnostic criteria has also been demonstrated by Zoccolillo *et al* (1986). In their study of 304 students at medical school in the United States, monthly scores on the Beck Depression Inventory (BDI; Beck *et al*, 1961) were obtained to select students for interview with the NIMH Diagnostic Interview Schedule (DIS; Robins *et al*, 1981). They found a 12% incidence rate (16% of women, 7% of men) of DSM-III major depressive disorder among students during the first two years of medical school together with a significant sex difference in rates. They concluded that depression was at least three times as common among medical students as in the general population.

The medical students in our study were little different from their US counterparts in terms of levels of reported emotional disturbance, almost 25% of the new intake in Edinburgh reporting experiencing the co-occurrence of at least five psychological symptoms during the study period.

Considerable recent research has been directed towards identifying those features of the medical-training environment that are associated with heightened levels of distress in students. Studies designed to look at student factors (e.g. Murphy *et al*, 1984; Notman *et al*, 1984; Lovallo *et al*, 1986; Sobal, 1986; Spiegel *et al*, 1986; Carmel & Bernstein, 1987; Rosenthal *et al*, 1987; Vitaliano *et al*, 1989) have relied mostly on self-report measures with assessments completed at intake and/or during the subsequent years of medical training. It is hoped that the availability of a 'guided' self-report measure of psychiatric symptom course, which is relatively efficient to administer, may facilitate the identification of factors that confer resilience or indicate the vulnerability of students to the largely inevitable stress associated with medical training. Findings concerning relationships between such factors and the changing mental health of the students as measured by the I-GHQ will be addressed in a future publication. Further development of the I-GHQ may involve, for example, giving due allowance in the scoring of symptom items to 'chronicity considerations' (e.g. Goodchild & Duncan-Jones, 1985; Surtees, 1987), and perhaps also to the choice of items and by asking subjects supplementary questions on impairment and help-seeking.

During the last few years there has been an increasing concern with many aspects of medical education, recently culminating in a world conference designed to identify agreed goals and strategies for the future. This conference concluded with a declaration in which specific aspects of medical training including curriculum and examination systems were identified as worthy of improvement (World Federation for Medical Education, 1988). This and other papers have shown that the transition to medical school is highly stressful and it would appear from other published work concerned with later stages of medical training that the educative process continues to be associated with heightened levels of emotional disturbance in the trainees. While it is to be hoped that efforts will be made towards achieving the ideals specified in the declaration, some consideration may also be given to the means of managing the very high levels of stress perhaps sometimes considered to be part of the 'rites of passage' of those undergoing medical training.

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References are given at the end of the following paper, pp. 692–693.

Appendix

Interval GHQ–interviewer guide notes

The purpose of this section of the interview is to obtain an overview of certain specific symptoms that each student may have experienced since their last interview. The assessment is based upon items from the General Health Questionnaire (GHQ).

Section format

Two subsets of items have been selected from the GHQ. One set of six items is concerned with anxiety symptoms

Scale no.	Item				Months			
		Score at time 1 (date)	Nov.	Dec.	Jan.	Feb.	Mar.	Score today (date)
					Anxiety set			
2	Lost much sleep over worry	1	—	1	2	—	—	2
3	Having restless and disturbed nights	2	—	—	—	—	—	2
14	Felt constantly under strain	1	—	—	—	1	3	4
19	Scared/panicky for no good reason	1	—	1	2	—	—	2
21	Everything getting 'on top' of you	2	—	—	—	2	1	1
28	Nervous and strung-up all the time	2	—	—	—	—	—	2
					Depression set			
22	Feeling unhappy and depressed	2	4	3	4	3	2	2
23	Losing confidence in yourself	2	4	—	—	4	2	2
24	Thinking of self as a worthless person	2	—	—	—	—	—	2
25	Felt that life is entirely hopeless	2	—	—	—	—	—	2
29	Felt that life isn't worth living	2	3	2	—	—	—	2
30	Couldn't do anything as nerves too bad	1	—	—	—	—	—	1

FIG. 2.

(the anxiety set), the other set of six items with depression related symptoms (the depression set). The 12 GHQ questions form the basis for this section of the interview. A separate interview coding form has been produced (Fig. 2). You will find the following information on each of these forms:

(a) the study code number associated with each student
(b) the two sets of GHQ items, divided according to whether they form part of the anxiety set or the depression set – each item has the scale number associated with the full 30-item questionnaire, as well as the GHQ question
(c) the actual scores for those items recorded by each student at the time of their initial interview is entered in the 'Score at' column: code 1 "Not at all", code 2 "No more than usual", code 3 "Rather more than usual", code 4 "Much more than usual"
(d) Note that the date of first interview also appears in the 'Score at' column
(e) The intervening months for each student are shown at the head of each blank column
(f) Note the 'Score today' column.

Procedure

On completion of the full GHQ, score the 12 items that appear on the form. Attention to the scale numbers on the form will help in searching for the items. Score each item 1, 2, 3 or 4 as above, and enter in the 'Score today' column.

Your aim now is to ascertain the student's symptom status over the interval between the ratings. While space is provided for monthly ratings the student does not have to be asked about how he/she was for each month. Instead probes should be used to determine change points which correspond to the GHQ scaled points. Monthly ratings can then be completed on the basis of identified change points. You should help the student to date change points, by asking such questions as "Was that in November?", "Did that happen before or after Christmas?", etc.

General

Examining the scores at initial interview will indicate symptom status at that time and that can be readily compared with current status for each symptom. Ask probe questions according to the pattern of ratings that each student presents. Do not assume that the interval is symptom free if both interview ratings are 1. In completing the forms, and where no change points are found, simply enter a line between ratings (which will be equal in score value). In general, enter a line covering times of equal ratings.

Fill in today's responses on the questionnaire

Some useful lead questions would be:

> "Now I want to look in slightly more detail at some of the items on the General Health Questionnaire."

"Last time you were interviewed, that is on (*give date*) you said about "Lost much sleep over worry" that this was (*give student's response*). How have things been since then?"

Now work through each symptom on the form probing to establish change points month by month. Be careful about using today's response, and very careful not to cross-examine the student about inconsistencies. Start each symptom off in the same way.

"When did you begin to feel better (or worse)?"

"Do you think that was a continuation of the way you were feeling at the time of the initial interview or a new period of (difficulty, depression, as appropriate) for you?"

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The Interval General Health Questionnaire and its Relationship with Psychiatric Disorder in Community and General Practice Samples

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This paper contrasts the usual scoring of the GHQ-30 with scoring based upon a criterion approach applied to only 12 of the GHQ items that takes account of the 'diagnostic significance' of the chosen items. Repeat assessments with this shorter form of the questionnaire (the I-GHQ), coupled with procedures to assess change, can be used to provide summary measures of symptom course in those research situations where available interview time precludes the use of more formal assessment methods.

The previous paper (this issue, pp. 679–693) describes a measure and procedure for documenting the presence and course over time of brief spells of anxiety and depression (and associated) symptoms. It explains how 12 items comprising the Interval General Health Questionnaire (I-GHQ) were extracted from the 30-item GHQ. While the selection of the 12 items comprising the I-GHQ was guided by diagnostic considerations (for inclusion with a criterion scoring method), the chosen items had previously been shown to have relatively superior sensitivity in relation to a severely ill group in the development of the GHQ. However, the final choice of the 12 items was empirically based. To underpin the results reported in the initial study, it is necessary to assess the relative performance in case identification of the chosen item subset in contrast to that of the longer form of the questionnaire (the GHQ-30) from which they were drawn.

While a comparison of the scales in one study is informative, the use of data from several study populations would be preferable. Ideally, these studies should be carried out in different settings and also with the application of different psychiatric case criteria. For the present study some attempt has been made to achieve this objective.

This paper is based upon the analysis of three data sets which together go some of the way towards meeting the above requirements for this investigation. As the method and design for each study has been described in some detail already, only an outline is given here.

Method

Study 1

This community study was undertaken in a geographically defined area of north Edinburgh. A random sample of 576 women aged 18–65 years selected from a total eligible population of about 44 000 (1981 census) completed an initial interview, and a subsample of those were re-interviewed six months and one year later. Details of the design and method of the initial survey can be found elsewhere (Dean *et al*, 1983; Surtees *et al*, 1983). Additional information concerning the follow-up interview method is also available (Surtees *et al*, 1986*a*,*b*). For the purposes of the present study, two particular groups of women were considered. The first of these, sample A, were those who met *either* Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978) *or* the diagnostic scheme allied to the Present State Examination (PSE; Wing *et al*, 1974; Wing & Sturt, 1978) for a current psychiatric disorder. Sample B comprised those women who, while free of psychiatric disorder at initial interview, reported having one or more of a set of putative vulnerability factors, *or* were included in a 40% random sample of those women who were both currently well and were found to have none of the factors. At each interview a semistructured interview, the Psychiatric Assessment Schedule (PAS), was completed (see Dean *et al*, 1983) and in addition (but only at each follow-up) the 30-item GHQ together with other measures was completed.

Study 2

This investigation was based in a 700 patient general practice in Verona (Italy). A total of 90 consecutive patients aged 14 years or over completed the Italian version of the GHQ-30 and within one week were interviewed by a psychiatrist with the Italian translation of the Clinical Interview Schedule (CIS; Goldberg *et al*, 1970; Fontanesi *et al*, 1985). Based upon the completed CIS ratings, a total weighted score was obtained, taking account both of reported symptoms and of observed abnormalities. Further details of the design of this study are given by Bellantuono *et al* (1987*a*,*b*).

Study 3

This Italian community study was undertaken in district N.4 of south Verona. A stratified probability sample yielded 208 households (representing 2.1% of the total number of households in the district) occupied by 585 residents. Data were finally obtained from 185 of the selected households which were occupied by a total of 534 residents, 454 being aged 14 years and over. For the purposes of the present analysis a random subsample of 88 families was selected. Among each of these families one subject aged at least 14 years of age was randomly selected and invited to complete the GHQ-30 along with other measures. Following the

TABLE I
Sensitivity and specificity of GHQ and I-GHQ subscores to case status

	Edinburgh community sample (RDC-determined cases)						*Verona study samples (CIS-determined cases)*	
	Sample A		*Sample B*		*Combined*		*General practice study (n=88)*	*Community study sub-sample (n=87)*
	F1 (n=61)	*F2 (n=59)*	*F1 (n=313)*	*F2 (n=283)*	*F1 (n=374)*	*F2 (n=342)*		
% with disorder[1]	55.7	40.7	7.0	3.5	15.0	9.9	37.5	26.4
GHQ ≥*5*								
sensitivity	79.4	50.0	86.4	100.0	82.1	64.7	87.9	78.3
specificity	74.1	71.4	85.6	81.7	84.6	80.5	70.9	76.6
Y coefficient[2]	0.51	0.12	0.71	0.87	0.66	0.46	0.60	0.53
I-GHQ								
All items ≥2								
sensitivity	76.5	58.3	95.5	90.0	83.9	67.6	90.9	82.6
specificity	70.4	65.7	82.8	76.9	81.8	75.6	70.9	60.9
Y coefficient[2]	0.44	0.15	0.80	0.65	0.65	0.42	0.65	0.43
Anxiety items ≥2								
sensitivity	70.6	58.3	95.5	90.0	80.4	67.6	90.9	73.9
specificity	81.5	74.3	85.2	78.0	84.9	77.6	76.4	70.3
Y coefficient[2]	0.50	0.28	0.82	0.66	0.65	0.45	0.69	0.41
Depression items >0								
sensitivity	70.6	54.2	90.9	90.0	78.6	64.7	69.7	69.6
specificity	74.1	68.6	89.3	86.8	88.1	84.7	72.7	70.3
Y coefficient[2]	0.41	0.14	0.80	0.75	0.67	0.51	0.40	0.37
Criterion basis[3]								
sensitivity	70.6	54.2	86.4	100.0	76.8	67.7	81.8	73.9
specificity	77.8	74.3	86.9	85.3	86.2	84.1	76.4	67.2
Y coefficient[2]	0.46	0.23	0.73	0.90	0.64	0.53	0.57	0.38

1. Nominal prevalence.
2. Based upon pseudo-Bayes estimation.
3. Cut-off point placed at 'least severe level'.

F1, follow-up approximately six months after initial interview; F2, follow-up approximately one year after initial interview.

completion of the questionnaire, and blind to its score, each subject was interviewed by two psychiatrists using the Italian version of the CIS. A total of five psychiatrists were involved in the CIS interviewing of the 88 subjects. All respondents were randomly assigned to pairs of psychiatrist raters. Each psychiatrist alternated between acting as interviewer and then as co-rater to complete their allocation of interviews. Further details of the sampling and design are given by Lattanzi *et al* (1988).

Analysis

Each data set was analysed in an identical way. Initially, the relationship between the conventionally scored GHQ-30 (reduced to a binary measure based upon the most usually applied cut-off point) and clinical psychiatric status was determined. Three measures were calculated: sensitivity (proportion of 'true' cases correctly identified), specificity (proportion of 'true' normals correctly identified), and *Y* (Yules' coefficient of colligation). While there is some dispute among statisticians concerning the latter measure's value over the kappa statistic (e.g. see Shrout *et al*, 1987), there have been persuasive arguments advanced in its favour (see Spitznagel & Helzer, 1985). In particular they have argued that differing values of kappa may be entirely due to variations in prevalence estimates but that use of the *Y* statistic minimises this base-rate problem. Further they have suggested that for studies of validity the *Y* statistic is completely independent of prevalence and relatively so in studies of reliability. The analyses to determine *Y* for this study were based upon smoothed pseudo-Bayes estimates of the cell probabilities (Bishop *et al*, 1975).

Sensitivity, specificity and *Y* were also calculated when the conventionally scored GHQ was replaced by four scores from the 12 items of the I-GHQ. The four measures were constructed as follows.

(a) An 'all-item score' was determined by summing all 12 items after conventional binary scoring and then imposing a cut-off point at ≥2.
(b) Two separate scores were determined after binary scoring of the six anxiety items and the six depression items, and cut-off scores set at ≥2 and >0 respectively.
(c) A criterion-based scoring of the I-GHQ was also undertaken identical to that in the previous paper. The scoring placed enhanced importance on two particular symptoms: from the anxiety set the item "Been feeling nervous and strung-up all the time" was the chosen key item, while from the depression set "Been feeling unhappy and depressed" was the key item. The presence of either or both of these symptoms, with or without any additional symptoms of the ten remaining (all rated according to the conventional GHQ binary scoring), was employed as an initial (general) criterion. Subsequently, three other criterion levels were imposed: none or one additional symptom of the ten possible (level 1), two or three additional symptoms (level 2) or four or more additional symptoms (level 3).

Results

The measures described above were initially calculated on data obtained from the Edinburgh study. Results based upon an analysis of the GHQ-30 on this sample have already been reported (Surtees, 1987) and for clarity they are partially reproduced in the top of Table I and in a format identical to that of the 1987 publication.

Also shown in Table I are the results obtained from examining the relationship between the four scores produced from the I-GHQ and RDC case status. These results are

TABLE II
The areas (and their variances) under the ROC curves determined according to conventional scoring of the full 30-item GHQ and of a selected subset of 12 items (I-GHQ)

Study centre and sample	*GHQ*		*I-GHQ*	
	Area	*Variance*	*Area*	*Variance*
Edinburgh				
Sample A				
F1 ($n=61$)	0.803	0.0038	0.819	0.0037
F2 ($n=59$)	0.698	0.0078	0.660	0.0087
Sample B				
F1 ($n=313$)	0.960	0.0004	0.962	0.0003
F2 ($n=283$)	0.952	0.0005	0.940	0.0010
Combined samples				
F1 ($n=374$)	0.907	0.0006	0.916	0.0005
F2 ($n=342$)	0.805	0.0026	0.797	0.0028
Verona				
General practice study sample ($n=88$)	0.863	0.0016	0.893	0.0013
Community study subsample ($n=87$)	0.823	0.0040	0.841	0.0034

F1, first follow-up; F2, second follow-up.

provided for the two study samples separately, after being combined, and at each of the follow-ups. Sample A are those women who met the RDC for a psychiatric disorder at initial interview, sample B those who were 'well' at that time. The indices are intended only to enable a comparison between the various scoring methods and not as precise estimates, given the deliberate sampling biases adopted in this study. (For a fuller discussion of the consequences for rate estimation of the study design see Surtees *et al*; 1986*a*). In general, the findings displayed in Table I and based upon the I-GHQ mirror those obtained and based upon the longer version of the questionnaire. Both forms of the GHQ performed relatively poorly on the 'recovery' case sample but particularly well on sample B. The results indicate no clear advantage or disadvantage of the I-GHQ over the GHQ-30.

Similar analyses were also completed on the two Italian study samples (missing data restricted the analysis to 88 general practice patients and 87 community subjects) (Table I). As with the Edinburgh sample, the overall results on the Verona samples indicate similarity in the performance of the longer and shorter forms of the questionnaire. It would appear, however, that the depression subset was marginally weaker than the anxiety subset in distinguishing CIS cases from non-cases.

An alternative analysis strategy for investigating the relative performance of a screening test in relation to a binary criterion is to use relative (or receiver) operating characteristic (ROC) analysis. This procedure enables all (or a selected subset) of cut-off points on a scale to be evaluated against the chosen criterion. To provide complementary results to those in Table I, the ROC method was applied to the Edinburgh and Verona data sets, enabling a more robust comparison of the GHQ-30 and its derivative in relation to the particular case criterion measures used in each study.

A computer program developed by Dorfman & Alf (1969), modified by Metz *et al* (1984; ROCFIT), was applied to the data. This program provides fitted (maximum-likelihood) estimates of both sensitivity and false-positive rate for all chosen scale cut-off points, and these can be plotted as ROC curves. The area under the fitted curve and its standard deviation are provided by this program. Table II displays the results and includes, to facilitate comparison, some findings already reported (Surtees, 1987) based on the GHQ-30 data from the Edinburgh sample. The results add weight to those already shown in Table I and confirm the broad equivalence in performance between the 12-item I-GHQ and the GHQ-30.

This result of course is not unexpected. There has already been considerable effort towards limiting the length of the GHQ at minimal cost in terms of its screening performance (Benjamin *et al*, 1982; Banks, 1983; Mari & Williams, 1985). However, the guiding principle in deriving the items for the I-GHQ was not screening ability, although this is a very important consideration, but that the items chosen should as far as possible provide a proxy indicator of diagnostic status based not on an aggregate scoring scheme but on similar principles to those being used in currently applied diagnostic schemes. A further, and equally important aim, was that the items should as far as possible be those most suitable for questions focused on *change* in symptom state.

The result of applying the criterion-based scoring scheme to the I-GHQ is shown in Table III (Edinburgh samples and Verona samples). Also shown is the nominal prevalence of psychiatric disorder in each subgroup. Discussion of the change (or 'fall-off') in these rates on the Edinburgh sample can be found elsewhere (see Surtees *et al*, 1986*a*; Surtees, 1987). The results in Table III show that the percentage of each sample meeting the highest anxiety criterion (level 3) was typically two to three times that of those meeting the highest criterion level of depression. A comparison of the percentage meeting level 3 (which requires at least one key symptom together with four or more others of the selected set) with the percentage with psychiatric disorder reveals the relative difficulty with which this level of the criterion is attained. In particular, of the 37 women (9.9%) out of the 374 combined Edinburgh samples who satisfied the requirements of level-3 anxiety, 23 reported all six anxiety-related symptoms. Of the 14 women (3.7%) from the same combined sample who met level-3 depression criteria, 10 reported all six depression-related symptoms The results obtained for the two Italian samples show the extent to which female prevalence exceeded that of the males.

An additional issue concerns the degree to which those attaining the various criterion levels satisfied the particular (but overall) psychiatric case requirements being used. The results in Table I showing estimates of sensitivity and specificity are essentially indicators of the validity of the various scoring methods. A measure of the potential of the criterion-scoring method for screening purposes is provided by the positive and negative predictive values (PPV and NPV) associated with the scoring scheme. These indices respectively indicate the probability that a screened positive will be a 'true case' or a screened negative will be a 'normal'. (A further discussion of these indices is given by Williams (1983) and Skuse & Williams (1984).)

PPV and NPV values were determined for the Edinburgh and Verona study samples based upon the I-GHQ combined anxiety/depression criterion-scoring scheme (Table IV). Also shown in Table IV is the nominal prevalence of disorder in each sample studied which showed a 16-fold variation according to the sample studied. Almost without exception, the PPV values associated with the criterion scoring scheme of the I-GHQ are greater than those with the GHQ-30 (as conventionally scored). The cost is a marginal reduction in negative predictive values. A fuller appreciation of the material displayed in Table IV may be gained by considering just one pair of results. Thus for the combined Edinburgh samples of 374 women at first follow-up (F1), 56 of 374 women (15.0%) (see Table I) fulfilled the requirements of the RDC for the range of psychiatric disorders assessed in this project. Imposing the I-GHQ criterion scoring with level 1 requirements suggests that 49.4% of women meeting the criterion would be 'cases' and 95.5% of those not meeting the criterion would not be RDC 'cases'. As can be seen from Table IV, this is about the same as for the conventionally scored GHQ. However, Table IV shows that increasing the (minimum) I-GHQ criterion symptom requirement increased the probability of respondents of being an RDC case; in this instance to 67.4%. The results also reveal that for the Edinburgh sample B and the two Verona samples PPVs increased with

TABLE III

Percentage of subjects meeting various I-GHQ criterion levels

	Edinburgh community sample of women (RDC-determined cases)						*Verona study samples (CIS-determined cases)*					
	Sample A		*Sample B*		*Combined*		*General practice study*			*Community study subsample*		
	F1 (n=61)	*F2* (n=59)	*F1* (n=313)	*F2* (n=283)	*F1* (n=374)	*F2* (n=342)	*All* (n=88)	*Males only* (n=27)	*Females only* (n=61)	*All* (n=87)	*Males only* (n=42)	*Females only* (n=45)
% with disorder	55.7	40.7	7.0	3.5	15.0	9.9	37.5	22.2	44.3	26.4	16.7	35.6
Anxiety criterion levels[1]												
3	26.2	16.9	6.7	6.4	9.9	8.2	14.8	7.4	18.0	13.8	7.1	20.0
2	13.1	6.8	2.9	6.4	4.5	6.4	18.2	18.5	18.0	9.2	4.8	13.3
1	3.3	1.7	2.9	0.7	2.9	0.9	8.0	7.4	8.2	6.9	7.1	6.7
0	57.4	74.6	87.5	86.6	82.6	84.5	59.1	66.7	55.7	70.1	81.0	60.0
Depression criterion levels[1]												
3	13.1	3.4	1.9	1.1	3.7	1.5	5.7	0.0	8.2	2.3	2.4	2.2
2	9.8	8.5	1.9	2.1	3.2	3.2	6.8	3.7	8.2	5.7	2.4	8.9
1	21.3	20.3	10.2	10.6	12.0	12.3	19.3	22.2	18.0	23.0	19.0	26.7
0	55.7	67.8	85.9	86.2	81.0	83.0	68.2	74.1	65.6	69.0	76.2	62.2
Combined anxiety/depression criterion levels[1]												
3	34.4	25.4	8.0	9.2	12.3	12.0	28.4	22.2	31.1	17.2	9.5	24.4
2	11.5	10.2	6.1	6.4	7.0	7.0	8.0	11.1	6.6	16.1	11.9	20.0
1	3.3	1.7	4.2	2.1	4.0	2.0	9.1	7.4	9.8	10.3	11.9	8.9
0	50.8	62.7	81.8	82.3	76.7	78.9	54.5	59.3	52.5	56.3	66.7	46.7

[1] See text for details.

F1, first follow-up; F2, second follow-up.

stricter criterion requirements and with little change in NPVs. The results suggest that under certain, perhaps special study situations, the criterion-scoring scheme of the GHQ items may have particular advantages over the conventionally scored GHQ-30.

Discussion

The GHQ is a widely used psychiatric screening instrument and since its original development has been evaluated in many settings and undergone intensive appraisal to establish 'improved' versions, either through alterations in length or item scoring (e.g. see Goldberg *et al*, 1976; Goldberg & Hillier, 1979; Tarnopolsky *et al*, 1979; Goodchild & Duncan-Jones, 1985; Bridges & Goldberg, 1986). The results of these endeavours are summarised by Goldberg & Williams (1988).

Why then should yet another form of the questionnaire be considered and what advantages could it possibly bring that have not already been achieved by earlier variants? Two points are of direct relevance to these questions. Firstly, the GHQ was primarily designed to be a psychiatric screening instrument and all the recommended forms of scoring have depended on an aggregate scoring method. To the authors' knowledge, criterion scoring has not been evaluated on the GHQ. This is rather surprising given the rationale adopted in currently applied research diagnostic schemes for fulfilling case criteria. It would seem worthwhile therefore to assess the value of imposing a relatively quick 'proxy criterion' through the self-report responses to these items in association with clinically assessed case status. This paper meets that objective and to the authors' knowledge is the first involving the GHQ to do so. The second point concerns the assessment of the course of the more minor episodes of psychiatric disorder. The selection of the particular subset of items used in this study was guided by both their broad relevance to the criteria used in diagnostic schemes for the more commonly occurring anxiety and depressive disorders and the relative ease with which change in the chosen items could be assessed over time using the principles of the LIFE methodology. The previous paper (this issue, pp. 679–686) has shown that those objectives have been achieved.

TABLE IV
Positive and negative predictive values (PPV and NPV) as percentages associated with the conventionally scored GHQ-30 and the I-GHQ after criterion scoring

Study centre and sample		*GHQ≥5*	*I-GHQ criterion scoring level* 3	≥2	≥1	0[1]
Edinburgh						
Sample A						
F1 ($n=61$)	PPV	79.4	81.0	82.1	80.0	(55.7)
	NPV	74.1	57.5	66.7	67.7	
F2 ($n=59$)	PPV	54.5	60.0	61.9	59.1	(40.7)
	NPV	67.6	65.9	71.1	70.3	
Sample B						
F1 ($n=313$)	PPV	31.3	56.0	40.9	33.3	(7.0)
	NPV	98.8	97.2	98.5	98.8	
F2 ($n=283$)	PPV	16.7	34.6	20.5	20.0	(3.5)
	NPV	100.0	99.6	99.6	100.0	
Combined samples						
F1 ($n=374$)	PPV	48.4	67.4	56.9	49.4	(15.0)
	NPV	95.4	92.4	95.0	95.5	
F2 ($n=342$)	PPV	26.8	43.9	33.9	31.9	(9.9)
	NPV	96.4	94.7	95.7	95.9	
Verona						
General practice study sample ($n=88$)	PPV	64.4	80.0	75.0	67.5	(37.5)
	NPV	90.7	79.4	83.9	87.5	
Community study subsample ($n=87$)	PPV	54.5	73.3	58.6	44.7	(26.4)
	NPV	90.7	83.3	89.7	87.8	

1. Represents the nominal prevalence of disorder (as a %) in each study.

So much for the stimulus behind undertaking this initiative, but do the results justify restricting use of the GHQ to the particular 12 items chosen and then imposing a criterion-scoring scheme? The answer to these questions must rest on the nature of the research envisaged. If longitudinal research is planned and there is very limited interview time available, then the I-GHQ, together with the procedure already described, may be found to be useful because of the greater detail provided on syndromal status over the chosen study period. For cross-sectional evaluations, the results of this study do not suggest that the I-GHQ should be the first choice but do indicate quite clearly that where a longer and more usual form of the GHQ has been applied, there may be distinct advantages of rescoring the subset of items to provide a criterion-based allocation of subjects. It is for future research to weigh these advantages further.

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Chronically Ill Psychiatric Patients in a District General Hospital Unit
A Survey and Two-Year Follow-Up in an Inner-London Health District

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A survey was undertaken at the end of 1984 of all patients occupying psychiatric beds for more than six months in an inner-London health district. Excluding those with senile dementia, 30 patients were identified. Two years later, a follow-up survey traced the original cohort and the accumulation of additional patients on the wards. The finding that the rate of discharge from the original group matches the accumulation of new patients into the survey suggests a need for new types of community provision within the district.

One of the most important debates in contemporary psychiatry, especially in Western Europe and North America, concerns the appropriate locus of care for the chronically mentally ill, with divided opinion about the feasibility of closing the large mental hospitals and replacing them with alternative, locally based provision, including care in the community. In Britain, surveys of populations in mental hospitals such as that of Levene *et al* (1985) have raised doubts about the capacity of health districts to manage their psychiatric services without the large hospitals.

Tooth & Brooke (1961) reported a trend of falling bed occupancy rates over 1954–59 for the long-stay population resident in mental illness hospitals in England and Wales at the end of 1954, such that that population would all have left hospital by 1975. However, they postulated the potential accumulation of a *new* group of long-stay patients at a rate of 89 per 100 000 population. Their paper was greeted optimistically by advocates of community care, who argued that improvements in treatment and rehabilitation programmes would prevent or diminish the development of the new long-stay population. In fact, new patients with long-term, severe disabilities have accumulated, as has been commented on by Wing & Hailey (1972), Measey & Smith (1973), Mann & Cree (1976), Todd *et al* (1976), Bewley *et al* (1981), Robertson (1981), McCreadie *et al* (1983), and Wykes (1983), among others. It has become clear that even with early treatment and active rehabilitation programmes, there remain substantial numbers of people (Robertson estimates current bed occupancy rates of around 40 per 100 000 population) with residual disabilities severe enough to require, in the long term, psychiatric services equivalent to those currently offered in hospitals.

Some health districts, however, are operating without access to large hospitals. Lawrence *et al* (1988) reported the experience of a district general hospital department formed as part of the Worcester Development Project. The authors found no evidence of accumulation of long-stay patients, but were cautious of generalising their findings to settings with greater demand for services and less social cohesion.

A recent development in the provision of long-term non-hospital-based care for the chronically ill is the concept of the 'hospital hostel', a separate facility for up to about 30 residents with 24-hour staff cover, but a more domestic setting. Such facilities are described by Garety & Morris (1984), Goldberg *et al* (1985), and Gibbons (1986).

City and Hackney Health District is an inner-London health authority providing all services within the district. There is no access to a large mental hospital. Despite extensive community resources, a group of patients was noted to be remaining on the acute wards for long periods, and this survey was commissioned to assess possible unmet needs within district provisions.

Psychiatric services in City and Hackney Health District

The district serves the City of London (population 5000) and the London Borough of Hackney, an inner-city borough with a population of just over 192 000, of whom 8.9% are aged over 65 (1981 census). The borough is high on most indices of social deprivation, with 20% unemployment, many single-parent families and heads of household born outside the UK, and poor housing stock (1981 census). Jarman (1984) describes a method of identifying underprivileged areas through examination of general practitioners' workloads, and on this index City and Hackney district is ranked third in England and Wales.

At the time of the initial survey, at the end of 1984, psychiatric services consisted of six psychiatric beds on a neurology ward at St Bartholomew's Hospital, 59 acute psychiatric beds at Hackney Hospital (a district general hospital), and 39 acute psychiatric beds at the German Hospital (a psychiatric hospital). There were two psychogeriatric assessment wards and three long-stay wards for elderly and severely mentally infirm patients. There were no other designated long-stay beds.

The provision of services within the district has changed dramatically over the last 20 years, with a well publicised commitment from the local division of psychiatry and the district health authority to provide a comprehensive service within the district using the district general hospitals as a focus. The ethos is of community care, providing continuing care for chronically disabled people in domestic settings.

In 1979, it became clear that the acute psychiatric wards were being occupied for long spells by chronically mentally disabled patients who did not need 24-hour medical and nursing care. They were considered to be inappropriately placed in hospital and in need of supported placements in the community. The Community Psychiatry Research Unit (CPRU) was established as part of St Bartholomew's Hospital Medical College, to study the needs of this group, and to facilitate the provision of services for them. In collaborations with the local authority and voluntary groups, CPRU has set up several housing projects including: independent living schemes with concerned neighbours on council estates, permanent accommodation for 15 people in a communal setting, a long-term home for ten medium-dependency clients with live-in staff, and a supported flat-share scheme. The district now has approximately 80 sheltered placements in the community, but at the time of the initial survey, none of these facilities catered for high-dependency clients.

Method

A census point of midnight on 31 December 1984 was chosen for the baseline survey. The names of all those patients admitted before 30 June 1984 were established from admission records and checked manually. Those with a clinical diagnosis of senile dementia were excluded. There were no patients under the age of 18. The 30 patients identified constituted a baseline population. A follow-up of this cohort was carried out exactly two years later (31 December 1986) to determine the whereabouts of the patients and to identify characteristics which differentiated those who had remained in hospital from those who had been discharged. The number of additional patients who had been in-patients for over six months on 31 December 1986 was also computed.

It was decided for two reasons that the criterion for duration of stay should be six rather than 12 months. It was the impression that patients who stayed more than six months were already placing a considerable burden on the acute service; we also wished to assess the risk of such people going on to become 'new long stay' as defined in other studies by a criterion of admission of 12 months. Other studies, such as that of Babiker (1980), support the validity of using six months as a cut-off point for defining chronicity, which also formed the basis of the National Institute for Mental Health (NIMH) Community Support Program guidelines (Tessler & Goldman, 1982). Simpson *et al* (1989) used six months as a definition of chronicity in a study comparing quality of life for patients in different facilities, both hospital and community.

An assessment schedule was administered to each patient in the cohort. This consisted of seven instruments designed to collect data on demographic characteristics, psychiatric history and physical health from case notes and care staff, social behaviour and environmental autonomy from care staff, and mental state and attitudes to services and social networks from patient interview. A full description of this schedule is being prepared for publication, and details are available from the authors on request.

Results

At baseline

Thirty patients fulfilled the criteria for entry into the survey. They were occupying just under 22% of the beds available on the acute and psychogeriatric assessment wards. There were eight men and 22 women, an unusual sex ratio in long-stay populations: in this group, the excess of women occurred after the age of 50 (Table I).

Personal data and psychiatric history

The patients in the study were, as expected, relatively unsupported by spouses or partners: only four of them were currently married or cohabiting, and 17 had never married; ten patients had a parent as next of kin and seven a sibling, four had no identifiable next of kin, while the other five patients had a child or other relative.

Ten patients, a third of the total, were without a permanent address, a common factor complicating rehabilitation in inner-city areas.

The length of current admission of the group had model and median values of between one and two years. Nine patients had been in hospital for less than a year and so by some conventions would not be included in a study of 'new long-stay' patients. These nine were compared with the other 21 for significant differences in sex ratio, age, permanent address, legal status, diagnosis, number of previous admissions, reason for remaining in hospital, attitude to leaving, physical health, mental state, and social behaviour. The only difference found between the two groups was in age, which was just significant at the 5% level (Wilcoxon, z score -1.99, $P=0.0462$), and it was therefore considered valid to analyse the results of the 30 patients together.

Only five patients were experiencing their first admission to a psychiatric ward, and the pattern of multiple admissions for the others demonstrates the 'revolving-door' phenomenon of repeated short admissions (Table I) with an emphasis on active rehabilitation and early discharge.

Only four patients were formally detained on the census date, although nine had been admitted under mental health legislation. The most common reason for admission was described as disturbed or aggressive behaviour (13 patients). Five had been unable to cope or were neglecting themselves, five were confused (in the lay sense of the word), one had been transferred from prison, and the rest had been transferred from medical wards or other psychiatric facilities.

TABLE I
Demographic data and diagnosis by sex

	Women (n=*22)*	*Men* (n=*8)*
Age: years		
range	33–81	21–72
median	58	37
Length of stay (current admission): months		
range	6–129	8–59
median	15	18
No. of previous admissions		
range	0–24	0–28
median	5	6
Total time in hospital before current admission: months		
range	0–88	0–>120
median	22	12
Diagnoses[1]		
Primary		
not made or non-psychiatric diagnosis	1	1
290 or 294 (organic psychoses)	0	4
295 or 297 (schizophreniform psychoses)	6	6
296 (affective psychoses)	0	9
300 (neurotic disorders)	0	1
315 or 317 (developmental disorders)	1	1
Secondary		
none	5	14
290	0	1
310 or 345 or 437 or 853 (other organic brain dysfunctions)	1	5
317 mild mental retardation	2	2

[1] ICD-9 diagnoses (World Health Organization, 1978).

Schizophrenia as a primary diagnosis was equally common in both sexes and was the commonest diagnosis in men (Table I). Affective psychosis was nearly as prevalent, being ascribed to nine patients, all women. Another striking finding was that 13 patients had gross organic impairment of brain function (two primary, four primary and secondary, seven only secondary to functional disorder). Two patients were taking no medication at all. Of the rest, eight were taking neuroleptics orally and nine intramuscularly, nine were on anti-Parkinsonian agents, ten on antidepressants, but only one was on lithium, and two were on benzodiazepines.

Physical health and activities

Physical health was assessed with a new instrument, the Physical Health Index. Eleven patients were taking medication for physical problems. Generally the physical health of the patients was worse among older patients. Sixteen patients had no physical disability at all, 12 had one disability (regarded as mild in eight patients, moderate in one, severe in three) and two patients had two disabilities (none severe). Eight patients suffered from degrees of what were termed 'critical disabilities', either incontinence, impaired mobility, blindness or deafness, which may have compromised their chances of being accepted for placements outside hospital. The level of care required for physical problems was established with the nursing staff and ranged from none at all (nine patients) to daily nursing care for ten. No patient was felt to require daily medical care for a physical problem. These assessments have great significance for planners, suggesting the need for high levels of nursing staff in any alternative provision being contemplated if these physical problems (as seems likely) prove to be unremitting.

The daytime activity of the patients was noted as occupational therapy for nine people and sheltered employment for one person. However, 20 patients were remaining on the ward unoccupied, suggesting that the activities being offered by the occupational therapy department were either unsuitable or unattractive for many of the most severely disabled.

Symptoms

Mental state assessment was performed using the Krawiecka scale (Krawiecka *et al*, 1977), a score of two or more on which indicates at least a moderate level of a particular symptom. The patients in the study exhibited high levels of all symptoms except hallucinations and thought disorder (Table II). There was an overlap between positive and negative symptoms and it was impossible to identify patient populations on this basis.

Social and cognitive functioning

Social performance was measured by the Social Behaviour

TABLE II
Numbers of patients assessed as having particular symptoms and items of behaviour

	Nos of patients
Symptoms[1]	
Depression	16
Anxiety	15
Delusions	18
Hallucinations	8
Thought disorder	6
Poverty of speech	17
Disorder of affect	18
Psychomotor retardation	13
Behaviour[2]	
Poor self-care	20
Unspecified behaviour	20
Inappropriate social contacts	16
Inappropriate conversation	15
Hostility	14
Unacceptable manners and habits	13
Underactivity	12
Attention seeking	10
Poor concentration	9
Overactivity	8
Depression	8
Taking initiative	7
Talking to self	5
Slowness	5
Posturing and mannerisms	5
Anxiety/panic	4
Incoherence of speech	4
Destructive behaviour	2
Sexual inappropriateness	1
Acting on delusions	1
Suicidal risk	0

1. Those scoring two or more for each item on the Krawiecka scale
2. Those scoring two or more for each item on the Social Behaviour Schedule.

Schedule (SBS; Sturt & Wykes, 1986). The SBS contains items relating to both deficits of normal behaviour and the presence of disturbed behaviour. A score of two indicates a moderate degree of disturbance, while three or more indicates a severe behavioural problem. The median number per patient of problems scoring at least two was six, while at the more severe level of disturbance (scores of three or four) the median number of problems per patient was four. Poor self-care and unspecified behaviour were the commonest problems identified, present to at least a moderate degree in 20 patients (Table II); the unspecified category includes behaviour such as fire risk from careless smoking and poor control of food intake. This high level of behavioural disturbance would contribute to the difficulty of finding alternative placements for some of the patients, particularly the elderly group, who, with less disruptive behaviour and self-care, might be acceptable for Part-III (local-authority) accommodation. None of the patients in the study was considered to be at risk of suicide.

Formal psychometric testing was not undertaken, but as a crude measure of cognitive function patients were asked their age, social circumstances before admission, and the duration of admission. A third of the group were unable to give their age and only seven people knew (to within 10% of the duration of admission) how long they had been in hospital.

The clinical staff on the wards were asked for their assessment of the reason for each patient remaining in hospital. Eighteen patients were awaiting alternative placement, three were considered to be institutionalised or unwilling to leave, and three had adverse social circumstances delaying discharge. Only six patients were considered by staff to be in need of hospital care on census date. There were no prognostic implications in their assessments. Of the three patients considered by staff to be unwilling to leave, one said on interview that she wished to remain in hospital, one expressed a strong desire to leave, and one was ambivalent.

Staff were also asked whether they felt that patients were realistic about future plans: 19 patients were felt to be either indifferent or to over- or underestimate their abilities. This in itself creates obstacles to enlisting the patient's co-operation in rehabilitation plans.

Patient attitudes

As part of a Patient Attitude Questionnaire (PAQ), patients were asked in an open-ended way about what they liked and disliked about living on the wards. Their responses were subsequently grouped together and reveal a rather indifferent attitude to their environment. Table III shows the categories of response to the questions.

Nearly half the patients (14) when interviewed expressed a strong desire to leave hospital, three had a qualified desire to leave, five patients were ambivalent, and two were vague, while only three people preferred to remain in hospital. The remaining three patients gave no response or did not know.

TABLE III
What patients liked and disliked about the ward

	Number of patients responding[1]	
	What do you like	*What do you dislike*
Don't know/no response	6	6
Nothing	11	6
Company (not care-givers)	3	6
Amenities	3	3
Permissiveness/restrictiveness	4	3
Delusional response	2	7
Care-givers	3	3
General approval/disapproval	2	2

1. More than one response can be chosen.

Hospital environment

The degree of autonomy available to the patients was measured on an Environmental Index (EI). Scores on the EI were compared with those obtained on long-stay wards of a local large hospital and found to be similar. The district has tried to adopt unrestrictive practices, for example with a policy that none of the wards should be locked. However, although the patients on wards at the district general hospital did not appear to be suffering greater restrictions than similar patients on long-stay wards, the results suggest that they are being disadvantaged by the presence of more disturbed individuals and are living in an inappropriate setting.

Social networks

An attempt was made to quantify the extent of the patients' social networks. Scores on the Social Network Schedule are easier to interpret by comparison with other groups, but a striking feature of the study sample was the general paucity of the networks and the particular lack of close and confiding relationships, with several patients unable to name a single confidant or person whom they would miss if they did not see them again. Two-thirds of the contacts mentioned were known because of psychiatric illness (i.e. staff or other patients) and of the social contacts nearly all were relatives. Follow-up will be needed in other settings to determine how much the limitations in social interaction are due to illness and how much to the effects of admission. The value of these findings is hard to assess as it was possible to complete the schedule comprehensively on only half the patients, and it was necessary to obtain additional data from staff in the other cases.

At follow-up

The flow chart (Fig. 1) indicates the movement of the patients over the two-year follow-up and their whereabouts on the second census date. Fifteen patients from the original cohort were in-patients on 31 December 1986. Ten of these had remained as in-patients continuously, and five had been discharged and readmitted. One patient was in a secure unit to which he had been transferred in March 1985 because of violence. Six were living in part-III accommodation; three were with relatives; two were in hostel accommodation, and two were in council flats (one in a community psychiatry research project). One, a 58-year-old woman with an organic psychosis, had died (from left ventricular failure). Thus of the original group, 22 (73%) were still in an institutional setting and seven were living outside hospital, only one of whom was living independently. The follow-up results for the continuous in-patient group, the discharged and readmitted group, and the discharged group are laid out in Table IV.

Seven of the nine patients in hospital for less than a year at baseline subsequently remained in hospital over a year, one patient was discharged after seven months, and one after 11 months. It seems that an admission of six months

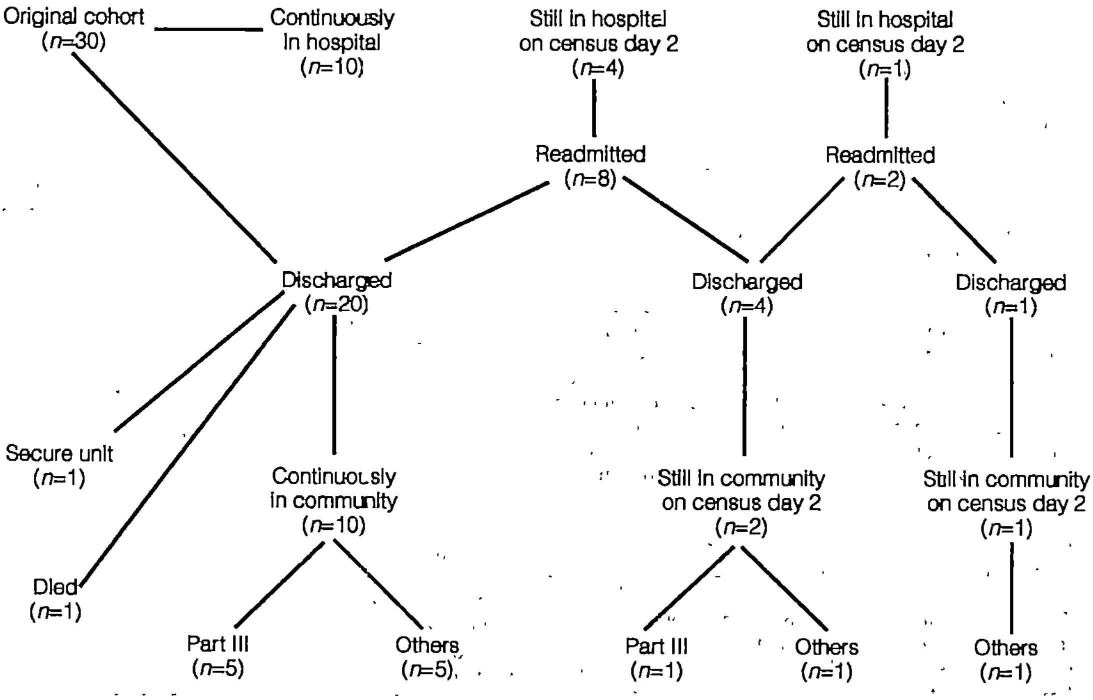
Original cohort (n=30)
Continuously in hospital (n=10)
Still in hospital on census day 2 (n=4)
Still in hospital on census day 2 (n=1)
Readmitted (n=8)
Readmitted (n=2)
Discharged (n=20)
Discharged (n=4)
Discharged (n=1)
Secure unit (n=1)
Continuously in community (n=10)
Still in community on census day 2 (n=2)
Still in community on census day 2 (n=1)
Died (n=1)
Part III (n=5)
Others (n=5)
Part III (n=1)
Others (n=1)
Others (n=1)

FIG. 1. Flow-chart showing movement of patients over two-year follow-up.

TABLE IV

Two-year follow-up of 30[1] long-stay patients, together with new long-stay patients identified at follow-up

	Continuous in-patients (n = 10)	*Discharged and readmitted (n = 5)*	*Discharged*		*New long-stay patients (n = 15)*
			Part III (n = 6)	*Other (n = 7)*	
No. of men	2	2	1	2	4
No. of women	8	3	5	5	11
Age: years					
range	27–83	35–71	58–82	23–64	25–80
median	61	60	71	50	50
Length of stay: months					
range	31–154	30–73	12–96	11–34	7–26
median	46.5	43	38	17	17
Total time in hospital: months					
range	0–88	17–51	0–23	0–67	0–45
median	6.5	40	6	23	3
No. of previous admissions					
range	0–25	9–28	0–8	0–24	0–39
median	5	15	3	9	6
Months in hospital to follow-up					
range	24	4–21	1–19	3–21	—
median	24	18	9	5	—
Diagnosis					
Schizophrenia	3	3	1	3	8
Affective psychosis	3	2	2	3	1
Organic psychosyndrome	3	—	1	1	4
Other	1	—	2	—	2
Secondary organic diagnosis	4	3	—	1	3
Total with organic diagnosis	7	3	1	2	7

[1] One patient was transferred to a regional secure unit and one died.

is a good predictor of an eventual length of stay greater than a year and we were thus identifying 'new long-stay patients' at an earlier stage of their illness.

Three patients not discharged between surveys had primary organic diagnoses and four had secondary organic diagnoses; three of the five patients discharged and readmitted had a secondary organic diagnosis. There was thus significant organic morbidity in these patients, who were also young, with a history of multiple short admissions (Table IV). They were unable to manage in the community even with support.

Of the 13 patients in the community at follow-up, seven had been discharged to relatives, hostels or council accommodation, and six to part-III homes. Overall, these patients were not unlike the discharged and readmitted group except that all of the first seven had relatives (who in five cases provided significant support), most had a home of their own, and they were amenable to the available support from community services. Indeed, two patients with organic problems had been discharged to caring relatives; a 57-year-old woman with multi-infarct dementia was now under the care of her husband, although she had been considered too disabled for Part-III accommodation, and a 41-year-old man with schizophrenia and mild mental handicap was living with his mother and attending a local day centre.

Five of the six patients in Part-III homes were free of any significant impairment of mental function, behavioural problems, or physical disability. The sixth was a 58-year-old woman with Korsakoff's psychosis and cerebral atherosclerosis. Given the chronicity of their problems and the lack of support from relatives, it is surprising that previous admissions had been so few in number and brief in duration.

In an attempt to identify possible predictors of successful discharge, the baseline results of the 11 patients who had remained continuously in hospital (including the patient transferred to a secure unit) were compared with those of the ten patients discharged and remaining in the community. The following variables were compared using χ^2 with Yates' correction: sex, age, presence of a permanent address, legal status at baseline, diagnosis at baseline, presence of a secondary diagnosis, length of stay, number of previous admissions, reason for remaining in hospital at baseline, attitude to leaving hospital, presence of disabilities and critical disabilities (the latter defined as those requiring daily nursing care), presence of symptoms and presence of behavioural problems. The Wilcoxon test was used for the distributions of age, length of stay, and number of previous admissions. There were no significant differences found between the two groups at the 5% level and it is probable that a larger sample would be needed to detect predictors of successful discharge.

Additional patients

On the second census date, 15 further in-patients were identified as having remained in hospital over six months (five for under a year). Their characteristics are shown in Table IV. These 'additional' patients were largely unsupported – 14 of them were single, widowed, divorced, or separated and seven were homeless. Six were formally detained on census date, although none had been compulsorily admitted. The most frequent reason for admission was disturbed or aggressive behaviour (eight cases); five had been unable to cope or were neglecting themselves, one was suicidal, and one had been transferred from another psychiatric unit. Five patients suffered from 'critical' disabilities. None of the group had a structured daytime activity. This group bears a striking resemblance to the original cohort.

Discussion

This study indicates that there has been an accumulation of long-stay patients in an inner-London health district since it stopped admissions to long-stay beds in a distant mental hospital. Over the two years of the study, the number of patients remaining in hospital for more than six months stayed constant, with half the original cohort leaving the hospital and being replaced by similar patients. The continued high bed usage by these patients has occurred despite relatively well developed community services and a commitment to community care. The results therefore differ from the Kidderminster study reported by Lawrence *et al* (1988) which showed no accumulation, but they are comparable with those of Mann & Cree (1976), who concluded that the 'new long-stay' patients would require hospital beds at a rate of 8–9 per 100 000, with a similar rate of hostel placements needed (using a 12-month admission as criterion of length of stay). The present study suggests a rate of 15 high-dependency places required per 100 000 population. If the nine patients who had been in hospital for less than a year on the first census date are discounted, the figure drops to 10.5 placements per 100 000. However, we would argue that there is good evidence to suggest that a duration of admission of six months is a more sensitive predictor of developing chronicity, and more useful to those planning services for such patients.

The patients in the study show many of the characteristics described in other studies of new long-stay and new chronic patients, namely high levels of dependence, homelessness, little family support, poor physical health, disruptive behaviour, and poorly controlled psychiatric symptoms. It is perhaps surprising that in an inner-city area with significant social deprivation and mobility, the rate of additional placements required is not higher than the national norm suggested by Mann & Cree. This suggests that the district has been successful in catering for all but the most dependent patients.

Although the 30 patients identified in this study represent the most disabled end of the spectrum of psychiatric illness, the staff caring for them on first census date felt that only six actually required hospital care. Of these, three had an affective psychosis, one schizophrenia, one pre-senile dementia, and one person had no diagnosis. All six manifested severe degrees of behavioural disturbance, three having eating problems (not anorexia nervosa), two refusing treatment, and one being a fire risk.

The 24 patients not in need of hospital care seem on inspection to fall into three discrete groups, although the numbers are insufficient for any statistical validation of differences between the groups.

(a) The 'organic' group. Five patients had a primary organic diagnosis, three of whom had no previous admissions and all of whom had a prognosis of either continuing static disability or of progressive deterioration in functioning. The problem of caring for the adult brain damaged and the role of psychiatry remains controversial and has been discussed by Newson-Smith (1983), among others.

Example Mrs A is a 56-year-old lady admitted six years previously for the first time with a diagnosis of Korsakoff's psychosis (non-alcoholic) and cerebral atherosclerosis. She has hypertensive retinopathy but no other localised physical signs. She seems to be cheerful and content on the ward and believes that she is in her childhood home. She becomes verbally abusive when reminded to do things and will take no interest in her self-care except to use the lavatory. Wandering is not a problem as she refuses to leave the ward. The prognosis is one of progressive deterioration. Staff feel that she would be better cared for in a more domestic setting but have been unable to find a place for her.

(b) The 'resistant-to-treatment' group. Ten patients had a primary diagnosis of functional psychosis (eight schizophrenia, two manic–depressive psychosis). Three of the schizophrenic patients and both manic–depressive patients were considered to be very resistant to treatment. Of the rest, three patients had a secondary diagnosis of mental handicap and two had suffered additional gross organic cerebral insult. The dependency needs of this younger chronic group are enhanced by multiple handicaps or lack of therapeutic response.

Example Miss B is a 34-year-old lady transferred back to the district 18 months previously, from a specialist behavioural unit. She has a diagnosis of hebephrenic schizophrenia and a history of six previous admissions totalling over seven years in hospital. Her illness is very resistant to treatment and she has remained persistently deluded and hallucinated. She has a history of epilepsy which is well controlled by medication. She talks mainly about her delusional beliefs; she is very rarely socially appropriate and often verbally abusive. She laughs and talks to herself and has several manneristic movements. She begs for cigarettes and picks up dog-ends to smoke. She expresses a vague but non-specific desire to leave hospital at some time in the future. The ward team are looking for more suitable provision outside hospital.

(c) The 'elderly' functionally mentally ill. Nine patients fell into this category and were either institutionalised, had physical problems, or were unacceptable for Part-III accommodation because of their marked symptoms and behavioural disturbance.

Example Miss C is a 73-year-old lady admitted four years previously with a diagnosis of endogenous depression, because she was unable to look after herself. She has had 14 previous admissions. Following a hip fracture she walks with a Zimmer frame. She continues to complain of depression and anxiety and has hypochondriacal delusions. She is very attention-seeking, agitated and restless on the ward, and the nursing staff have great difficulty in persuading her to eat. She is sometimes incontinent of urine and faeces. Staff feel that she underestimates her ability and is scared of appearing well in case she is discharged from hospital. The patient strongly agrees that she wants to stay in hospital.

At two-year follow-up, 15 of the original cohort of 30 patients were in hospital, ten continuously and five discharged and readmitted. The continuous group had a median length of admission of three years at first census date and had severe organic and behavioural problems throughout admission. The five with failed community placements had a history of multiple short admissions and significant organic pathology. Of the 15 patients, three had been considered in need of hospital care on first census date; of the remaining 12, three belonged to group (a), six to group (b), and three to group (c).

Although we were unable to demonstrate any significant differences in baseline data between those patients who remained continuously in hospital and those who were subsequently discharged and stayed in the community, analysis was complicated by the discharge and subsequent readmission of eight patients, of whom five then stayed in hospital. The continuous in-patients and the group with failed community placements both showed high levels of organic psychosyndromes and, in retrospect, it would have been desirable to attempt a comprehensive screening of cognitive function, as the presence of organic brain disease may play a major part in predicting long-stay status. It seems that in this district, the psychiatric services are assuming an active role in the care of the adult brain damaged.

Conclusion

The original study and follow-up confirm the gaps in community provision for particular types of patient: the functionally psychotic with additional handicaps, the adult brain damaged, and the elderly with functional mental illness and behavioural problems. The patients in each of these categories were 'high dependency' and may well be most appropriately catered for in the types of long-term facility described under the 'hospital hostel' model. Significantly different facilities will be needed for each of the three types of patient. Residential and day-care plans for these groups will need to accommodate intensive staffing, and this requirement is likely to be long term since, although physical disability was not especially predominant at the time of the census, the prognosis for the organic and elderly patients is one of increasing physical and psychological dependency. There are considerable capital and revenue implications in the full provision of suitably adapted and adequately staffed care facilities for these very dependent patients.

Perhaps the most important finding of the study is that the number of long-stay patients appears to reach a steady state, with outflow balancing intake. This study was conducted in a very deprived area and the results may not have general relevance. Nevertheless, two important findings are not limited by the social context: the use of a six-month admission predictor of chronic mental illness and high dependency, and the importance of organic brain disease in determining long-term disability. Future studies may benefit from more extensive use of formal testing of cognitive function.

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Vasopressin in Chronic Psychiatric Patients with Primary Polydipsia

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Twelve chronic in-patients with primary polydipsia were studied, during free drinking and after fasting, by concurrent measurements of plasma AVP, serum sodium and osmolality, and urine volume, AVP, osmolality, and creatinine. A majority of the patients showed inappropriately high levels of AVP: plasma AVP estimations demonstrated that seven had Type I SIADH and two had Type II SIADH. Urinary AVP estimations confirmed inappropriately raised AVP in seven of the subjects tested, and there was a significant agreement between the plasma and urine diagnoses. Although able to concentrate their urine in response to fluid deprivation, the patients showed a decreased renal sensitivity to AVP. Despite the mitigating effect of decreased renal sensitivity to AVP, the SIADH seen in these patients appears to contribute to the development of water intoxication caused by polydipsia.

In 1963, Hobson & English suggested that the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) caused the water retention seen in a polydipsic patient suffering from chronic schizophrenia who had become intoxicated with water. Since then, there has been considerable interest in the role that arginine vasopressin (AVP), the antidiuretic hormone, plays in patients with self-induced water intoxication. Plasma AVP has been examined in studies conducted with varying degrees of control of relevant variables in about 50 polydipsic patients, most of whom had suffered episodes of water intoxication. The results of these investigations have been varied: although abnormalities in AVP secretion have frequently been documented, no single common abnormality has been found. No paper focusing primarily on AVP in patients with this disorder has included urinary AVP estimations, but two papers included such data on one and two patients respectively with compulsive water drinking; these did not report plasma AVP estimations. In the present study, psychiatric patients with primary polydipsia were examined during periods of free drinking and fluid deprivation, and plasma and urinary AVP levels were determined concurrently under controlled conditions.

The first serum AVP measurement in such patients was reported by Raskind *et al* (1975): a high level was found in a hyponatraemic case suffering from psychotic depression. Although the AVP estimation was not repeated, the patient went on to show a normal response to a water-loading test, and hence a transient SIADH could be inferred. Fowler *et al* (1977) also described a single case of apparently transient SIADH in a schizophrenic polydipsic patient whose serum AVP, estimated during periods of normal water excretion, proved to be normal.

Later studies involved larger numbers of patients and sometimes a greater degree of control of potentially confounding variables. It was only realised quite recently that many psychiatric patients with primary polydipsia undergo regular and rapid fluctuations in the degree to which they are water loaded; earlier studies at times assumed relatively constant hydration, and samples were not always obtained concurrently when it would have been appropriate. In those studies in which plasma AVP was examined, posture was controlled only in the study of Vieweg *et al* (1988*a*), but even here, it is not mentioned how long the patients were sitting before venipuncture. Smoking was controlled in only a few of the studies. Seizures cause AVP release (Aminoff *et al*, 1984), but no paper to date has specified the periods elapsed since the patients' most recent seizures. It is known that urinary tract abnormalities are common in these patients, but again, no paper to date has taken into account the fact that bladder emptying may be incomplete. The presence or absence of orthostatic hypotension is often omitted. Finally, details of the method of the AVP analysis are often missing, and sometimes even the type of analysis is omitted. These methodological points provide the framework for the following brief review of the literature.

Vieweg *et al* (1984*a*, 1984*b*, 1985*a*, 1987, 1988*a*) have described patients with inappropriately high AVP levels and have concluded that these were cases of Type I SIADH, in which there is no relationship between plasma osmolality and plasma AVP (Robertson, 1979). Ragavan *et al* (1984) concluded that many patients with polydipsia and hyponatraemia have a reset osmostat for AVP secretion (Type II SIADH), supporting the conclusion of Hariprasad *et al* (1980), whose work did not include AVP estimations. Inoue *et al* (1985) described two patients with elevated plasma AVP levels at the time of hyponatraemia-induced convulsions, but normal AVP levels later. They questioned whether the transient episodes of elevated AVP were secondary to hyponatraemic encephalopathy. Bourgeois *et al* (1985) reported inappropriately high AVP levels in two patients who had suffered convulsions resulting from hyponatraemia; in one of these, subsequent testing showed a reset osmostat. Hamazoe *et al* (1986) described SIADH in three of 16 polydipsic schizophrenic patients, as well as relatively low urine osmolality for serum AVP, inadequate suppression of serum AVP following a standard water load, and an inadequate excretion of the water load in those patients with polydipsia of more than five years' duration. The most comprehensive and best controlled study to date was by Goldman *et al* (1988). Although posture was not mentioned, smoking was forbidden from the night before the study until its completion the next afternoon. The patients were given a standard water load, followed by an infusion of hypertonic saline. There was a decreased osmotic threshold for AVP release in the polydipsic patients compared with a non-polydipsic control group matched for psychiatric diagnosis (all but one suffered from schizophrenia). In addition, the polydipsics showed an apparently enhanced renal sensitivity to low levels of AVP. The defect in water excretion could not be attributed to inadequate suppression of AVP release.

In regard to urinary AVP excretion, Merkelbach *et al* (1975) described a patient with compulsive water drinking who was able to increase urinary AVP concentration to 27 pmol/l during a period of fluid deprivation, and who excreted 42 pmol in 22 hours, fluid intake being permitted only at the beginning of this period. Khokhar *et al* (1978) described two patients with compulsive water drinking who showed a normal relationship between AVP excretion (43–75 fmol/min) and urine osmolality (140–750 mosm/kg) during 21–31 hours of fluid restriction.

Method

Patients

Chronic in-patients with polydipsia were identified by a survey at Kingston Psychiatric Hospital. Twelve patients with primary polydipsia, comprising nine men and three women, consented to undergo investigations. According to DSM–III–R criteria (American Psychiatric Association, 1987), eight men and two women suffered from chronic schizophrenia; one of these men had also suffered a head injury after the onset of the schizophrenia. One man suffered from alcoholic dementia and one woman from chronic atypical psychosis. The mean age was 41.3 years (range 30–54) and mean duration of polydipsia, defined as the number of years since hyponatraemia, hyposthenuria (urine specific gravity ≤ 1.003), obvious polydipsia or any other complication was first noted, 9.6 years (range 2–29).

Ten patients were receiving neuroleptics and two of these were also receiving lithium carbonate. The patients on lithium were included for their intrinsic interest. Lithium has a number of potentially confounding effects, which necessitate separate treatment of these patients' results where appropriate in the data analysis. The mean neuroleptic dose, in chlorpromazine (CPZ) equivalents (Wyatt, 1976), was 1386 mg/day. No patient was receiving an anticonvulsant or a diuretic, and none had received electroconvulsive therapy in the year preceding the study. All but one smoked. Complications related to polydipsia which have been described by Vieweg *et al* (1984*b*) and Delva *et al* (1988, 1989) were common. Seven patients had suffered hyponatraemic seizures and other serious consequences including osteopenia and pathological fractures, urinary tract abnormalities, and hernias; two had experienced episodes of hyponatraemia without serious effects; two had shown hyposthenuria (urine specific gravity ≤ 1.003) without more serious consequences; and one, in spite of obvious polydipsia (as reflected by polyuria of over 8l/24h), had no history of hyponatraemia, hyposthenuria as defined above, or other consequences, although his urine specific gravity had been measured as low as 1.004. In the five patients without serious consequences, the polydipsia had shorter durations (mean 3.0 years, range 2–5). Because of the implementation of a weight-monitoring procedure (Delva & Crammer, 1988), no patient had suffered a seizure in the year preceding the study.

There was no evidence of systemic illness unrelated to the polydipsia in 10 patients; one schizophrenic man suffered from hypertension (treated with captopril and nifedipine) and the patient with alcoholic dementia had become diabetic secondary to pancreatitis and received insulin. During states of normal hydration, no patient showed significant abnormalities in haemoglobin, white cell count, serum electrolytes, magnesium, total calcium, phosphate, urea nitrogen, creatinine, protein, albumin, cholesterol, triglycerides, liver function tests, uric acid or thyroxine. Blood sugar after fasting for 10–12 hours was normal in all patients except the one with diabetes, whose level was only mildly elevated at 8.0 mmol/l. Plasma cortisols at 08.00 h and 22.00 h were normal in 10 patients; the levels were abnormally high at both times in the patient with a history of head injury and at 22.00 h in the one with alcoholic dementia. Creatinine clearance was normal to high in all patients because of generally low serum creatinine levels.

No patient suffered from significant orthostatic hypotension; the mean blood pressures (BP) were: lying–systolic 126 (s.d. 16)/diastolic 80 (s.d. 10); sitting – 122 (s.d. 19)/83 (s.d. 12); and standing – 121 (s.d. 17)/82 (s.d. 11). These figures include the hypertensive patient, whose BP was 146/98 (lying).

Procedure

The study extended over several days. During the first 24 hours ('day 1'), each patient was under constant observation by nursing staff. Body weight was measured at 06.00 h with bladder empty and the patient was allowed to drink freely up to 10% over this weight; weight and urine volume were recorded hourly. The urine for each four-hour period was pooled and samples taken for estimations of osmolality, creatinine, and AVP; the sample for AVP analysis was frozen at −20°C. Blood was taken at the midpoint of each four-hour urine collection during the daytime, at 08.00, 12.00, 16.00, and 20.00 h; the patient was not allowed to smoke for one hour and had to sit for half an hour before venipuncture, for which a tourniquet was used. Samples of blood for AVP analysis were collected in lithium heparin tubes and were placed immediately in melting ice after the blood was drawn; these were quickly spun and the plasma frozen at −20°C. Samples of blood for estimations of sodium and osmolality were collected in unheparinised tubes; these were also quickly spun, and the serum frozen for subsequent analysis.

On a subsequent day, fluid intake was restricted to 2 litres (i.e. to establish a state of normal hydration) and then nothing was permitted by mouth from 20.00 h until 08.00 h the next day ('fasting'). Weight was determined and urine collected from 06.00 h to 08.00 h, and blood was taken at 08.00 h.

Following this, blood was obtained over a period of days to ensure that plasma AVP was estimated over a range of serum osmolalities, body weight being used as a guide (Delva & Crammer, 1988).

The measurements of sodium, creatinine and osmolality were performed in the hospital laboratories by established methods: sodium by an ion-sensitive electrode, creatinine by the method of Rossignol et al (1984), and osmolality by freezing point depression. Frozen samples of plasma and urine were analysed for AVP by radioimmunoassay (Williams et al, 1985).

Analysis

A repeated measures analysis of variance, paired *t*-tests, and Pearson product-moment correlations were employed for the statistical analysis. One-tailed *t*-tests were used to test the statistical significance of the correlations. Regression procedures were used to develop equations to describe some of the phenomena. Urine AVP concentrations were expressed as 'less than 2.5' or 'less than 5' pmol/l 15 times in 82 assays. On these occasions, the assay provides an upper bound estimate; the values lie in the tail of the frequency distribution of the observations somewhere between zero and the upper bound value. The tail might be expected to have an approximately triangular shape, as extreme values (in this case very small) tend to be much rarer than those around the median. If this reasoning is sound, we determined that for the upper bound values given in the present data, about half would lie below the value of 0.7 times the ceiling (i.e. 1.8 and 3.5 pmol/l respectively for the 2.5 and 5 pmol/l ceilings), while the other half would lie above these values. Thus, for these upper bound observations, a value of 0.7 times the upper bound was chosen as likely to be an unbiased estimate of the true value.

Results

In spite of the intrusive nature of the protocol, polydipsia was present on Day 1 in most of the patients, as shown by the polyuria in Table 1. Urinary creatinine excretion was actually greater than the predicted values of 0.16 mmol/kg/day or 10.6 mmol/day for men and 0.11 mmol/kg/day or 6.6 mmol/day for women, indicating that urine collections were complete. Osmolar excretion was within normal limits as defined by Warhol et al (1965) in all but one patient. Urinary AVP excretion in normals depends on their state of hydration; a number of investigators have determined AVP

TABLE I

Measurements over 24 hours of free drinking in 9 male (M) and 3 female (F) polydipsic patients compared with those of normals

		Polydipsics		
Variable	*Normals*	*Mean*	*s.d.*	*Range*
Urine volume: l	<3	M 7.3	3.5	3.2–13.6
		F 3.7	1.2	2.4–4.6
Urinary creatinine excretion: mmol/day	M 9–18[1]	13.0	2.8	10.3–19.0
	F 7–16	10.3	2.2	7.7–11.6
Urinary osmolar excretion: mosm/day	M 767–1628[2]	1000	248	595–1383
	F 433–1146	765	107	643–809
Urinary AVP excretion: pmol/day	25–130	M 50.0	15.8	25.1–72.4
	(normal hydration)[3]	F 63.2	11.0	52.3–72.0
Cigarettes smoked		M 22.0	3.6	17–29
		F 15.3	13.6	0–26

1 Normal limits as defined by Faulkner & King (1976).
2. Normal limits as defined by Warhol et al (1965).
3. See text for discussion of normal values.

excretion under hydrated, normal and dehydrated conditions, usually investigating the rate over a few hours (see Claybaugh & Sato, 1985 for a review, and Seckl *et al*, 1986). Miller & Moses (1971, 1972) and Merkelbach *et al* (1975) reported 24-hour excretion rates, and the normal range in Table I is that of the former. This range includes all but one of the reported mean hourly excretion rates multiplied by 24, the exception being that of Khokhar *et al* (1978) who corrected their results for recovery losses, thus increasing the values. Only Merkelbach *et al* (1975) provided mean values for men (66 pmol/day) and women (32 pmol/day) separately, although they did not provide ranges. Values for hydrated individuals are generally lower than normal, water loading often reducing urinary AVP excretion to undetectable levels. The mean value under water-loaded conditions for all the papers cited by Claybaugh & Sato (1985) and Seckl *et al* (1986) was 32 pmol/day as compared to 83 pmol/day under normal conditions. Thus, considering the hydrated state of our polydipsic patients, the mean urinary AVP excretion was somewhat greater than would be expected.

A repeated measure analysis of variance was used to compare means of the different variables for each time or period during Day 1; when there was reliable change over the 24 hours, the mean measurement for each time or period was compared with the fasting value by paired *t*-tests, and when there was no reliable change, overall means for Day 1 were compared with the fasting values. Table II and Fig. 1 show the results of plasma and urine tests during Day 1 and after fasting. There was no significant fluctuation in creatinine excretion rate during Day 1.

Trend analysis showed that the regression of weight (w) on time (t) during Day 1 was adequately described by a linear function: $w = 64.9 + 0.49t$, where $t = 1$ for 08.00 h, 2 for 12.00 h, 3 for 16.00 h and 4 for 20.00 h ($r = 0.99$). The relationships between urine excretion rate (uexr) and time (t) and between urine osmolality (uosm) and time (t) were quadratic in form, being best expressed by the functions: $uexr = 1.2 + 4.5t - 0.7t^2$ (multiple $R = 0.91$) and $uosm = 328 - 100t + 16t^2$ (multiple $R = 0.96$), where $t = 1$ for the period 06.00–10.00 h, 2 for 10.00–14.00 h (Fig. 1).

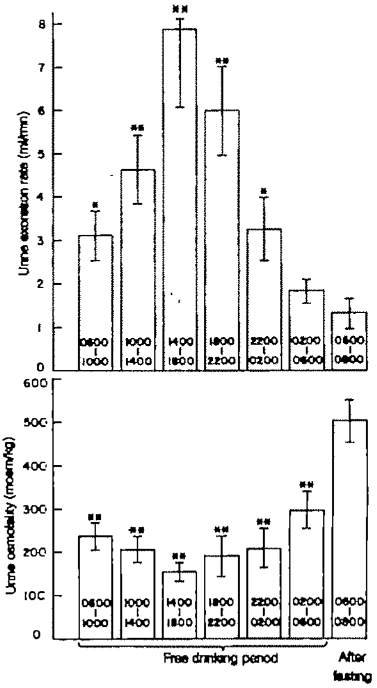

FIG. 1 Urine excretion rate and urine osmolality in 12 polydipsic patients, over 24 hours of free drinking divided into four-hour periods, and after fasting for 10–12 hours following the establishment of a state of normal hydration (bars indicate standard errors, and significant differences between free drinking and fasting values are indicated: *$P<0.05$; **$P<0.01$).

Figure 2 shows the relationship between serum osmolality and plasma AVP during the entire study period. The norma range, shown by the shaded area, is taken from Lightman & Everitt (1986) and represents values from normal subjects using the same assay as employed in the present study. A considerable degree of abnormality was present. When al

TABLE II

Measurements during free drinking and after fasting in 12 polydipsic patients

	Day 1				
	Period or time	*Value during free drinking (s.e.m.)*	*d.f.*	*t-test*	*Value at 08.00 h after fasting (s.e.m.)*
Body weight: kg	08.00	65.4 (2.1)	11	2.85**	64.7 (2.2)
	12.00	65.8 (2.1)	11	3.12**	
	16.00	66.5 (2.2)	11	4.76**	
	20.00	66.8 (2.3)	11	4.93**	
Mean serum osmolality: mosm/kg	08.00–20.00	279.8 (2.1)	11	6.46**	290.0 (1.5)
Mean serum sodium: mmol/l	08.00–20.00	137.2 (1.3)	11	3.88**	141.5 (0.7)
Mean plasma AVP: pmol/l	08.00–20.00	2.4 (1.1)	10	2.03*	3.0 (0.5)
Mean urine AVP: pmol/l	over 24 h	15.9 (3.9)	10	3.02**	52.2 (10.1)
Mean urine AVP:creatinine ratio: pmol:mmol	over 24 h	4.5 (0.6)	10	2.25*	7.6 (1.2)

*$P<0.05$; **$P<0.01$ (paired one-tailed tests comparing mean values for each time or period on Day 1 with fasting value).

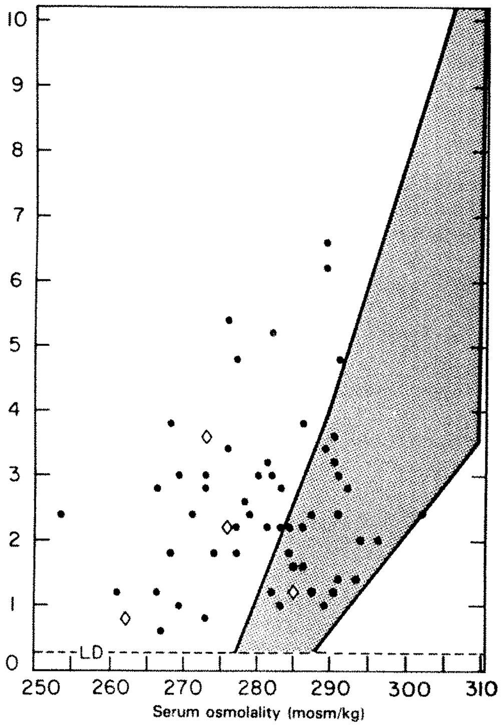

FIG. 2 Concurrent serum osmolality and plasma AVP in 12 polydipsic patients (normal range indicated by the shaded area (Lightman & Everitt, 1986); LD = limit of detection of the assay; ● = individual data points; ◇ = two superimposed data points).

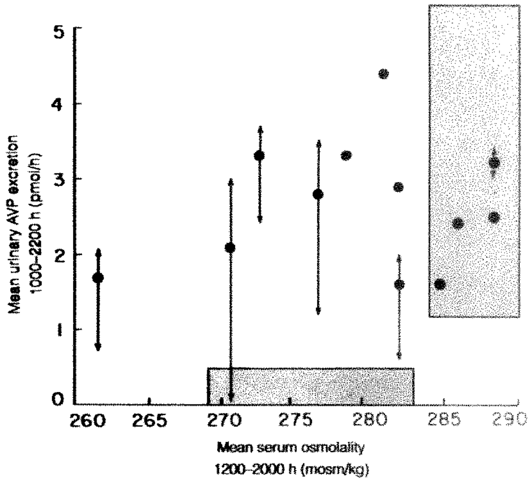

FIG. 3 Concurrent mean serum osmolality and mean urinary AVP excretion during maximum hydration on the free drinking day in 12 polydipsic patients (arrows indicate ranges of possible values for six patients; shaded areas indicate normal ranges (Miller & Moses, 1972)).

data points were analysed together, without regard to differences between patients, the correlation between osmolality and AVP was not reliably different from zero ($r = 0.17$; d.f. = 63; NS). The x-intercept of the linear regression equation was 169 mosm/kg and the slope 0.02 pmol/l per mosm/kg. When correlations were calculated separately for each individual, *z*-transformed, averaged, and converted back to correlations, the mean *r* rose to 0.36 (t (11) = 2.74; $P < 0.01$), indicating heterogeneity in the osmotic set point for AVP release (i.e. different intercepts for the regression lines) or in the amount of AVP induction resulting from a unit change in serum osmolality (i.e. different slopes for the regression lines). An examination of individual patients showed that of the three patients whose AVP levels fell into the normal range, all were schizophrenic, two were female, and the male was treated with lithium. Of those whose AVP levels were abnormal, two showed statistically significant correlations between serum osmolality and plasma AVP ($r = 0.93$ in each patient; $P < 0.01$) and could be described as having Type II SIADH (reset osmostat), but seven showed non-significant correlations between the two variables and thus suffered from Type I SIADH.

Figure 3 shows the relationship between mean Day 1 serum osmolality during the period of maximum hydration (12.00–20.00 h) and concurrent (10.00–22.00 h) mean urinary AVP excretion. This period was chosen to minimise the effect of incomplete bladder emptying (see Discussion). The normal ranges are taken from Miller & Moses (1972) and are indicated by the shaded areas. The data points include some estimates for concentrations of 'less than 2.5' and 'less than 5' pmol/l; ranges are also provided in these cases, which included between one and three ceiling values. Again, considerable abnormality was present. The correlation between the two variables was low at 0.18 (NS) as was that between mean Day 1 plasma AVP (12.00–20.00 h) and concurrent urinary AVP excretion ($r = 0.25$; NS). It is apparent that only those patients whose mean serum osmolality fell into the normal range excreted normal quantities of AVP. When patients were categorised as manifesting normal or abnormal plasma or urinary levels of AVP, compared with serum osmolality, seven were abnormal by both measures, three normal by both measures (including one receiving lithium), one abnormal by plasma but normal by urine, and one (receiving lithium) abnormal by plasma and unclassifiable by urine, as his urine AVP excretion rate could have been either normal or abnormal. The agreement between these two categorisations is significant by Fisher's exact test ($P < 0.05$) and supports the validity of the assessment of urine AVP.

Figure 4 shows the relationship between plasma AVP and concurrent urine osmolality from 10.00 h to 22.00 h on Day 1 and while fasting. The normal range is taken from Goldman *et al* (1988) and covers the relationship between plasma AVP and urine osmolality up to a plasma AVP of about 2.5 pmol/l. Again, the 06.00–10.00 h period on Day 1 was excluded in order to minimise the effect of incomplete bladder emptying, but for this analysis, the results were not averaged. It is apparent that there is a lack of

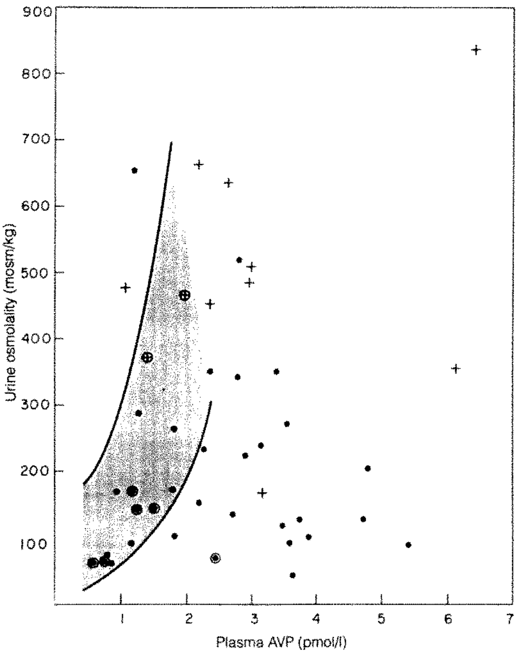

FIG. 4 Concurrent urine osmolality and plasma AVP in 12 polydipsic patients (the shaded area indicates the normal range, Goldman *et al*, 1988), up to a plasma AVP of about 2.5 pmol/l; ● = data point from four-hour collections on Day 1 (10.00–22.00 h); + = data point from fasting period (06.00–08.00 h); ⊙ ⊕ = lithium treated patients).

response by the kidney to circulating AVP and that this is not explained by lithium in the two patients who received this drug.

Table III shows mean correlations for complete concurrent data in the 12 patients. The direction of each correlation is as would be expected.

Evaluation of the effects of smoking yielded the following results: there was no correlation between the number of cigarettes smoked and the amount of urine excreted ($r = 0.28$; NS) but a significant correlation between cigarettes smoked and mean urine osmolality ($r = -0.64$; $P < 0.05$). The correlation between amount smoked and mean daytime plasma AVP was 0.38 (NS), and that between amount smoked and mean urine AVP: creatinine ratio only 0.13 (NS). There were no apparent effects, by correlational analysis, of neuroleptic dose, expressed as CPZ equivalents per kilogram body weight, on any of the other variables measured.

Discussion

Although able to respond to fluid deprivation and consequent relative dehydration with increased vasopressin secretion and concentration of the urine, many of our patients did show inappropriate AVP release (SIADH), as evidenced by a lack of suppression of plasma and urine AVP levels during the periods of maximum hydration, and abnormal relationships between plasma and urine AVP and serum osmolality. In addition, they showed a decrease in renal sensitivity to these inappropriately high levels of AVP.

Our findings indicate heterogeneity among chronic psychiatric patients with primary polydipsia as to presence and subtype of SIADH and accord best, in general, with those of Vieweg *et al* (1984*a*, 1984*b*, 1985, 1987, 1988*a*), in that Type I SIADH was the most common form of abnormality. They differ in

TABLE III
Mean correlations for complete concurrent data in 12 polydipsic patients

	Serum osmolality	*Plasma AVP*	*Body weight*	*Urine creatinine*	*Urine osmolality*	*Urine AVP : creatinine ratio*	*Urine excretion rate : ml/min*
Serum sodium : mmol/l	0.88	0.41	−0.79	0.66	0.75	0.64	−0.52
serum osmolality : mosm/kg		0.36	−0.69	0.74	0.81	0.58	−0.55
Plasma AVP : pmol/l			−0.61	0.39	0.73	0.60	−0.39
Body weight : kg				−0.73	−0.81	−0.58	0.74
Urine creatinine : mmol/l					0.98	0.48	−0.73
Urine osmolality : mosm/kg						0.63	−0.71
Urine AVP : creatinine ratio: pmol/mmol							−0.28

some respects from those of Goldman *et al* (1988) whose study showed a lower than normal osmotic threshold for AVP release, but demonstrated that AVP release was almost completely suppressed below a plasma osmolality of about 279 mosm/kg, and completely suppressed below about 274 mosm/kg. In addition, our finding of decreased renal sensitivity to AVP is at variance with their finding of increased sensitivity. The different results of the two studies may simply reflect patient heterogeneity, or be consequences of the several differences in experimental design: firstly, the water load was administered over a period of 15 minutes in Goldman *et al*'s study (20 ml/kg) whereas in ours, the water loading was self-administered, much greater, and given over a much longer period; secondly, smoking was completely forbidden in their study, but was permitted at specific times in ours; thirdly, urine was collected over periods of 30 minutes in their study, while in ours, either two- or four-hour collections were made.

The present study was designed to allow the evaluation of AVP secretion in our patients under conditions which conformed as closely as possible to their daily routine. Since most of these patients are habitual smokers, it was judged least disturbing, both to their subjective states and behaviour, and to their neuroendocrine function, to allow our patients to smoke during the period of evaluation, although not during the hour before venipuncture. Even in habitual smokers, smoking may cause an acute rise in plasma AVP; the AVP level peaks approximately 10–20 min after finishing a cigarette but returns to baseline by 50 min after doing this (Rowe *et al*, 1980; Chiodera *et al*, 1987). In the investigation of Dietz *et al* (1984), a single cigarette did not raise plasma AVP levels immediately after smoking; several in close succession were required to raise plasma AVP, and again the effect was short-lived. Thus, we cannot attribute the inappropriately high plasma AVP levels seen in our patients to smoking. It remains unclear to what extent smoking contributes to the development of water intoxication, although a causal relationship was suggested by Chin *et al* (1976) and Blum (1984). These investigators did not measure AVP levels in their patients, but conducted water-loading tests comparing water excretion during abstinence from smoking with that during heavy smoking (15 cigarettes in two-and-a-half hours and 20 cigarettes in three hours respectively). Smoking at these rates greatly exceeds normal consumption, yet would be required for a sustained elevation of plasma AVP in normal habitual smokers. In such smokers, there is a close relationship between plasma nicotine and plasma AVP; Pomerleau *et al* (1983) found a correlation of 0.985 between integrated plasma nicotine and plasma AVP. However, Vieweg *et al* (1986) failed to find any significant correlation between plasma AVP and serum nicotine in patients such as ours, and concluded that the brain abnormalities causing inappropriate AVP release in these patients were sufficient to overshadow any effects that nicotine might have. As far as the effect of smoking on urinary excretion of AVP is concerned, the lack of correlation between smoking and urine AVP : creatinine ratio in our patients is evidence against smoking-induced AVP secretion being responsible for the abnormally high urine AVP levels.

A lack of complete bladder emptying can be a problem in investigations like this. Unless bladder catheterisation is employed, it is likely that at least some of the patients have partially full bladders at the end of each collection period. This fact must be taken into account when data are interpreted. Incomplete bladder emptying may have accounted for the apparent increased renal sensitivity to AVP found by Goldman *et al* (1988). In our study, the relationship between plasma AVP and urinary osmolality was examined over periods during which there was minimal fluctuation of urine osmolality and hence the influence of incomplete bladder emptying was minimised. The finding of decreased renal sensitivity to AVP is in keeping with the decreased renal concentrating ability seen in normal subjects subjected to prolonged high fluid intake (De Wardener & Herxheimer, 1957; Habener *et al*, 1964), and is in agreement with the finding of Hamazoe *et al* (1986). The decreased renal sensitivity to AVP in our patients was not explained by lithium treatment, which was a possible factor in Vieweg *et al*'s (1988*a*) patients who showed decreased sensitivity to AVP but were all treated with lithium. In addition to its renal effects, lithium has also been found to have effects on the osmoregulation of AVP secretion (Gold *et al*, 1983) but does not cause SIADH.

The plasma AVP levels seen in our patients appear adequate to cause water retention. In normal subjects, the full range of renal concentrating and diluting capacity is covered by a ten-fold change in plasma vasopressin concentration, from 0.46 to 4.6 pmol/l (Robertson *et al*, 1976). Our patients showed a considerable increase in urine osmolality, after fluid deprivation, with a mean plasma AVP level of 3.0 pmol/l. Thus, although apparently mitigated by a decreased renal sensitivity to AVP, the levels observed during over-hydration appear sufficient to cause water retention, thus exacerbating the effect of excessive fluid intake.

Raskind *et al* (1987) found that the antipsychotic drugs haloperidol and chlorpromazine did not directly stimulate vasopressin release in either normal controls or in acute schizophrenic patients, but could indirectly stimulate its release via baroreceptor reflex mechanisms if drug-induced hypotension occurred. Since our patients did not manifest such hypotension, the neuroleptic therapy that most of them were receiving is unlikely to have been the cause of the SIADH. However, the conjecture of Goldman *et al* (1988), whose patients also received neuroleptics, that abnormalities in water metabolism may in part reflect an idiosyncratic response to neuroleptic therapy, cannot be excluded in the present study.

The purpose of our investigation was to determine whether abnormalities in AVP secretion existed in these patients, rather than to elucidate the causes of the polydipsia or of any possible abnormality in AVP secretion. The effects of the polydipsia, exacerbated in many cases by SIADH, are many. For the brain, there is a constantly changing ionic environment and episodic brain swelling. Any abnormalities in nervous-system function discovered in these patients may, therefore, be results, rather than causes, of the polydipsia; thus the SIADH in our patients may, at different times in the development of the syndrome, be effect or contributing cause or both.

Both excessive fluid intake and SIADH contribute to the development of hyponatraemia in these patients. Thus treatment may be aimed at the reduction of fluid intake and, in those with SIADH, at the reduction in release or effect of AVP. At present, the most conservative treatment is water restriction, using body weight as a guide to the state of hydration. Water restriction may be used if polydipsia is one of the symptoms of an acute psychotic episode and discontinued if neuroleptic or mood-stabilising treatment relieves the polydipsia; it may be used over the long term if symptomatic polydipsia persists despite optimal pharmacotherapy. Over the last decade, such patients have undergone therapeutic trials with drugs that affect drinking, the release of AVP, or the action of the hormone on the kidney. These drugs include propranolol (Shevitz *et al*, 1980; Kathol *et al*, 1986), demeclocycline (Nixon *et al*, 1982; Khamnei, 1984; Goldman & Luchins, 1985; Vieweg *et al*, 1988*b*), lithium (Vieweg *et al*, 1985*a*), sodium chloride (Vieweg *et al*, 1985*b*, 1988*c*), lithium in combination with diphenylhydantoin (Vieweg *et al*, 1988*c*), and captopril (Kathol *et al*, 1986; Lawson *et al*, 1988). With the exception of the study by Nixon *et al* (1982), which employed placebo-control and double-blind methods, all of these trials were open, and the numbers of patients small (total number of cases for all trials was 44). Outcome measures generally included morning or random serum sodium and urine concentration and sometimes frequency of episodes of hyponatraemia or seizures. From the limited amount of work done, there is some indication that demeclocycline and the combination of lithium and diphenylhydantoin may be helpful, but each form of therapy may also be toxic, and possibly unsuitable for long-term use. There is less evidence that captopril is useful and propranolol has only been evaluated in three patients. Additional therapeutic trials are required, both to evaluate further those drugs already tried, and to assess the value of other medications, including those already employed in the treatment of SIADH, such as urea which is a safe and effective osmotic diuretic (Decaux & Genette, 1981) as well as those suggested by basic research, such as clonidine, a potent antidipsic in animal models of thirst (Fregley *et al*, 1984). Finally, behavioural methods (McNally *et al*, 1988) require further evaluation.

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The Dexamethasone Suppression Test in Anorexia Nervosa

The Influence of Weight, Depression, Adrenocorticotrophic Hormone and Dexamethasone

ISAAC SCHWEITZER, GEORGE I. SZMUKLER, KAY P. MAGUIRE, LEONARD C. HARRISON, VIRGINIA TUCKWELL and BRIAN M. DAVIES

When 20 female anorexic in-patients were investigated with weekly DSTs, 10 had an abnormal result at initial testing. There was no identifiable relationship between severity of weight loss and DST status; % ideal body weight was no different between suppressors and non-suppressors. There was no consistent relationship between normalisation of the DST response and weight gain. Depressive symptoms were common, with half the patients scoring 20 or more on the HRSD. Plasma ACTH concentrations before and after the DST were normal. There was a significant negative correlation between plasma dexamethasone concentrations and pre- and post-dexamethasone plasma cortisol concentrations.

There is substantial evidence of abnormalities in the functioning of the hypothalamic–pituitary–adrenal (HPA) axis in the low-weight phase of anorexia nervosa (Weiner, 1985). Anorexic patients have an increased cortisol production rate (Walsh *et al*, 1978), as well as elevated 24-hour concentrations of plasma cortisol (Walsh *et al*, 1987). Boyar *et al* (1977) found an increased half-life and a reduced metabolic clearance rate of cortisol in anorexia nervosa. In addition, Gerner & Gwirtsman (1981) reported non-suppression of cortisol after administration of dexamethasone in 18 of 19 anorexic patients.

It has been generally assumed that the hypercortisolism in anorexia nervosa is a consequence of the low weight itself, and that this abnormality is corrected with a return to normal weight, but previous investigations of HPA-axis functioning in anorexia nervosa have studied patients either in the low-weight phase alone or in the low-weight and weight-corrected phases of their illness: no previous studies have serially monitored HPA-axis functioning during the course of treatment. To investigate the relationship between weight change and the HPA axis, our anorexic patients received weekly weighings and dexamethasone suppression tests (DSTs) during their stay in hospital. We chose the DST as a measure of HPA-axis functioning because of its simplicity and ease of application.

As the incidence of depressive symptoms is high in anorexia nervosa (Kirstein, 1981; Gershon *et al*, 1984), it has been suggested that HPA-axis dysfunction might be a consequence of the depression. Supporting this hypothesis are studies of other neuroendocrine abnormalities that are common to anorexia nervosa and depression: specifically, lowered levels of urinary 3-methoxy-4-hydroxy-phenylglycol (Halmi *et al*, 1978; Gwirtsman & Gerner, 1981), and a blunted response of thyroid-stimulating hormone and exaggerated responses of growth hormone to stimulation by thyrotrophin-releasing hormone (Macaron *et al*, 1978; Gold *et al*, 1981–81) are found in both conditions. In addition, several systematic family studies demonstrate a higher than expected prevalence of affective disorder in the relatives of patients with anorexia nervosa (Gershon *et al*, 1984; Biederman *et al*, 1985). To investigate the possible relationship of anorexia nervosa and depression further, patients were carefully evaluated for the presence of depressive symptoms and severity of depression was assessed weekly.

As recent work has shown dexamethasone kinetics may influence the DST response (Holsboer *et al*, 1984; Johnson *et al*, 1984; Maguire *et al*, 1987), dexamethasone concentrations were measured at the initial DST. In addition, to further define underlying pathophysiological mechanisms of HPA-axis dysfunction in anorexia nervosa, concentrations of adrenocorticotrophic hormone (ACTH) were also studied.

Method

Over three years, patients admitted to the Psychiatric Unit of the Royal Melbourne Hospital (RMH) meeting DSM–III (American Psychiatric Association, 1980) diagnostic criteria for anorexia nervosa, were studied. All patients were initially in the low-weight phase of their illness, being less than 80% of their ideal body weight (IBW) (Metropolitan Life Insurance Co., 1959; mid-point for medium frame). Patients had a detailed physical examination and laboratory screening tests to exclude serious physical disorder.

Eighteen normal female volunteers who were in good physical and mental health participated in the study. The volunteers were recruited from among staff of the RMH and University of Melbourne. Height, weight, blood pressure and heart rate were measured. Personal details including age, sex, alcohol and tobacco consumption, menstrual status and current medication were obtained.

No subject who met the medical exclusion criteria detailed by Carroll *et al* (1981*a*) was included in the project. In addition, subjects receiving carbamazepine were excluded as it is known to cause false positive results (Privetera *et al*, 1982). Written informed consent was obtained from all subjects.

Between the third and seventh day in hospital, all patients received a standard DST as specified by Carroll *et al* (1981*a*) with 1 mg dexamethasone being administered at 2300 h and blood samples being taken the same and following day at 1600 h. The DST was repeated at approximately weekly intervals during the patient's hospital stay. The normal volunteers received the standard DST as an out-patient procedure.

All blood samples were analysed for plasma cortisol. Plasma ACTH and dexamethasone concentrations were measured at the initial DST only. Plasma cortisol was measured by the radioimmunoassay in routine use in the Department of Biochemistry, RMH (Amerlex, Amersham, UK). The interassay coefficient of variation (CV) was 5% at 180 nmol/l, 530 nmol/l and at 1220 nmol/l. Plasma ACTH was measured in unextracted plasma in the Endocrine Laboratory, RMH, using a commercially available kit produced by CIS-Sorin from the Australian Atomic Energy Commission. The ACTH assay had a sensitivity of 4.0 pmol/l with a CV of ±12% at 13 pmol/l and ±6% at 32 pmol/l. Plasma dexamethasone was measured by radioimmunoassay using unextracted plasma. ^{3}H-dexamethasone (specific activity 50 Ci/mmol) was obtained from NEN Research Products, Boston, USA. The antiserum was a gift from Professor Vecsei, Heidelberg, Germany. Its cross-reactivity to cortisol was approximately 0.02%. The CV for the dexamethasone assay was ±12% at 0.5 ng/ml, ±15% at 1.0 ng/ml, and ±12% at 2.0 ng/ml.

All patients received the 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1965) and the self-rating Carroll Rating Scale for Depression (CRSD; Carroll *et al*, 1981*b*) assessments during the first week of admission. The HRSD and CRSD were repeated at weekly intervals during each patient's hospital stay. The assessor was blind to the laboratory DST results. All normal volunteers completed the CRSD.

All patients, dressed in a gown only, were weighed and their height measured at admission. Weekly weighings were continued during the patients' hospital stay. All normal subjects were carefully questioned concerning recent weight change and their weight and height were recorded.

A post-DST plasma cortisol concentration of >187 nmol/l defined non-suppression. This value was the 95th percentile in a previous study of our cortisol assay with 100 normal volunteers (Tiller *et al*, 1988).

Statistical analyses were performed using SPSSx programs (SPSSx Inc., 1986). Tests for significance were parametric or non-parametric tests where appropriate. Variables were tested for normality using the Kolmogorov–Smirnov one-sample test. For comparisons between groups, independent *t*-tests or non-parametric Mann–Whitney *U* tests were used. For comparisons within groups, dependent *t*-tests or non-parametric Wilcoxon tests were used. Significance levels were set at $P<0.05$. Relationships between variables were tested by their Pearson product–moment correlations or by Spearman correlations for non-parametric data.

Results

The 20 female anorexic patients were younger and weighed significantly less than the normal controls, and their pre-DST and post-DST plasma cortisol concentrations were significantly higher (Table I). Of the 20 anorexic patients, 10 were non-suppressors at their initial DST. All the normal volunteers were suppressors.

TABLE I
Comparison of results in anorexia nervosa patients and normal controls

	Anorexic patients		*Controls*		t *or* U	P
Mean age (s.d.): years	21.1	(5.1)	27.0	(4.7)	$t=3.63$	$P<0.001$
Mean % IBW (s.d.)	70.0	(8.4)	101	(14)	$t=9.09$	$P<0.001$
Mean pre-DST cortisol (s.d.): nmol/l	431	(156)	288	(103)	$t=3.24$	$P=0.001$
Mean post-DST cortisol (s.d.): nmol/l	201	(178)	40	(19)	$t=3.82$	$P=0.001$
Mean dexamethasone (s.d.): ng/ml	0.67	(0.45)	0.82	(0.61)	$t=0.833$	NS
Median pre-DST ACTH (range): pmol/l	7.3	(4.4–44.4)	7.2	(4.4–16.4)	Mann–Whitney $U=146$	NS
Median post-DST ACTH (range): pmol/l	5.2	(4.4–158)	4.4	(4.4–15)	Mann–Whitney $U=198$	NS

TABLE II
Comparison of results in anorexic patients according to DST status at initial testing

Suppressors (n = *10)*	*Non-suppressors* (n = *10)*	t *or* U *value*	P	
Mean % IBW (s.d.)	70.2 (10.2)	69.7 (6.7)	$t=0.06$	NS
Mean HRSD score (s.d.)	16.3 (8.1)	20.2 (6.4)	$t=1.19$	NS
Mean pre-DST cortisol (s.d.): nmol/l	327 (115)	536 (120)	$t=3.78$	$P=0.002$
Mean post-DST cortisol (s.d.): nmol/l	54 (27)	349 (113)	$t=6.88$	$P=0.000$
Mean dexamethasone (s.d.): ng/ml	0.92 (0.48)	0.44 (0.25)	$t=2.77$	$P=0.013$
Median pre-DST ACTG (range): pmol/l	9.1 (4.4–44.4)	5.5 (4.4–38)	Mann–Whitney $U=41$	NS
Median post-DST ACTH (range): pmol/l	4.4 (4.4–158)	6.7 (4.4–10.4)	Mann–Whitney $U=40$	NS

Ten patients were less than 70% of their IBW. There was no apparent relationship between severity of weight loss and evidence of HPA-axis dysfunction (Table II). In addition, there was no correlation between post-DST cortisol in the initial DST and %IBW. There were three patients who were less than 60% of their IBW at admission but only one was a non-suppressor.

Of the ten non-suppressor patients, seven had become suppressors by discharge. There was no apparent relationship between normalisation of the DST response and increase in body weight. At the week of normalisation of the DST response, three patients had made significant gains in weight (5.5 kg, 11.2 kg, 12.2 kg), three patients had gained only 0.9 kg, 1.4 kg and 3.0 kg, and the weight of one was unchanged.

There was no apparent relationship between failure to normalise DST response and lack of weight gain. Two patients whose DST response had not normalised at the time of discharge had gained 11.2 kg (reaching 69%IBW) and 12.8 kg (reaching 89%IBW). (The remaining non-suppressor patient was in hospital for less than three weeks and had failed to improve.)

Depressive symptoms were very common among the anorexic patients, 17 scoring 10 or higher on the HRSD. Ten patients had an HRSD of 20 or more. However, there was no significant correlation between post-DST cortisol in the initial DST and HRSD ($r=0.24$, NS) or between %IBW and HRSD ($r=0.02$, NS), and there was no significant difference in mean HRSD between suppressors and non-suppressors (Table II).

There was a trend for HRSD score to diminish during the stay in hospital. At the time of discharge, nine patients had an HRSD score over ten, and only two an HRSD score over 20. Only four of the patients had received specific antidepressant therapy. There was no clear relationship between normalisation of the DST response and remission of depressive symptoms.

Six of the 20 anorexic patients had evidence of bulimia, five of whom were non-suppressors; however, there was no significant relationship between DST status and presence of bulimia ($\chi^2=2.14$, $P>0.1$).

There was no difference in ACTH concentrations before or after dexamethasone between the anorexic and normal group (Table I). In addition, pre- and post-DST ACTH concentrations, between suppressor and non-suppressor patients, were not significantly different at the initial DST (Table II).

There was no significant difference in dexamethasone concentrations between anorexic patients and normal volunteers (Table I). However, dexamethasone concentrations were significantly higher in suppressor than non-suppressor patients (Table II). In addition, in the anorexic patients there was a significant negative correlation between cortisol and dexamethasone concentrations both before and after initial DST ($r=-0.64$, $P=0.004$, and $r=-0.60$, $P=0.003$, respectively). There was no significant correlation between dexamethasone concentrations and %IBW, nor between dexamethasone and HRSD.

Discussion

In this study half the anorexic patients in the low-weight phase of their illness were found to have evidence of HPA-axis dysfunction; this is consistent with previous work. In a previous report of eight anorexic patients our data suggested DST non-suppression occurred when patients were less than 70%IBW (Schweitzer *et al*, 1986). However, in this study with increased patient numbers, our results indicate that DST non-suppression in anorexia nervosa is not a simple function of severity of low weight; pre- and post-DST plasma cortisol concentrations did not correlate with %IBW. In addition, normalisation of the DST response was not always associated with significant weight gain.

These results suggest that more than one mechanism is responsible for an abnormal DST in anorexia. The effect of acute weight loss, rather than the degree of weight loss itself, may be a factor. There is evidence that fasting in normal individuals may result in DST non-suppression (Berger *et al*, 1984; Mullen *et al*, 1986). It is, therefore, possible that anorexics who have experienced a recent acute loss in weight will be initially non-suppressors and will become suppressors as a consequence of weight stabilisation rather than weight gain. This indicates that further research must carefully distinguish acute weight loss from severity of weight loss when investigating anorexia nervosa.

There were two patients who were less than 60%IBW but who were DST suppressors. One of these patients became a non-suppressor after gaining weight. These two patients suggest that HPA-axis functioning may be affected in a different manner in the severely wasted state. A possible explanation is that with more severe weight loss, cortisol production may be diminished, only then to be increased with a gain in weight. Further investigation with careful attention to the severity of weight loss in anorexics is required.

Basal and post-DST ACTH concentrations were found to be no different in anorexic patients compared with normal controls, including patients who had an abnormal DST. We know of no previous studies of ACTH in relation to the DST in anorexia nervosa. Our findings are consistent, however, with those of two previous studies of stimulation of corticotrophin-releasing factor (Gold *et al*, 1986; Hotta *et al*, 1986) which found high cortisol but normal basal plasma ACTH concentrations in anorexic patients. Also, these studies found marked blunting in the plasma ACTH response; these investigators interpreted their findings as suggesting an abnormality of the HPA axis at the level of the hypothalamus or higher centres in the central nervous system.

Our finding of normal concentrations of ACTH is consistent with the findings of ACTH-stimulation studies in anorexia nervosa which suggest adrenal hyper-responsiveness (Danowski *et al*, 1972; Warren & Van de Wiele, 1973). These studies suggest the presence of another factor(s) which enhances the sensitivity of the adrenal cortex to ACTH. This factor might be another hormone which is known to influence cortisol secretion, such as β-endorphin (Taylor *et al*, 1983). This is clearly in need of further investigation. In addition, as cortisol and ACTH have a pulsatile secretory pattern, there would be considerable interest to study anorexic patients with multiple and frequent sampling of these hormones, perhaps over 24 hours.

Depressive symptoms were frequent, with half the patients having an HRSD of 20 or more. Correction of HPA-axis abnormalities and remission of depression tended to occur during the course of the hospital stay but this was not consistently associated with weight gain. Furthermore, patients with evidence of HPA-axis dysfunction were no more severely depressed, as measured by HRSD, than those without such dysfunction. Although these data argue against depression being a primary factor causing HPA-axis dysfunction in anorexia nervosa, they do not rule out the possibility that depression may have a role in an abnormal DST in some anorexic patients.

Our findings indicate inadequate dexamethasone concentrations may be a factor in causing an abnormal DST in some anorexic patients and the role of dexamethasone kinetics in anorexia nervosa requires further investigation. Anorexics who were non-suppressors were found to have significantly lower concentrations of dexamethasone than suppressors. Furthermore, there was a significant negative correlation between pre-DST plasma cortisol and dexamethasone concentrations and between post-DST plasma cortisol and dexamethasone concentrations. There have been several recent studies with similar findings in depressed subjects (Holsboer *et al*, 1984; Johnson *et al*, 1984; Maguire *et al*, 1987) but the significance of dexamethasone concentrations remains uncertain. Poland *et al* (1987) suggest that approximately one-third of depressed subjects might be misclassified as non-suppressors owing to inadequate dexamethasone concentrations. As we did not measure dexamethasone concentrations serially, the influence of weight change on dexamethasone requires further clarification. At the initial DST there was no correlation between %IBW and dexamethasone concentrations. However, it is possible that with improvement and weight gain, alterations in dexamethasone kinetics may be a significant factor in the changing DST status of patients.

This study suggests that the pathophysiology of HPA-axis dysfunction in anorexia nervosa is more complex than has been previously thought; furthermore, the nature of the abnormality may be a function of several mechanisms operating at different levels of the HPA axis.

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The Efficacy of Lithium in Prophylaxis of Unipolar Depression
Evidence from its Discontinuation

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Forty unipolar patients satisfying DSM-III criteria for major depression who discontinued lithium therapy were retrospectively compared with 105 similar patients who continued the drug and served as a control group. The time to readmission from starting lithium was compared while both groups were still on lithium, and after discontinuation in one group and further continuation in the control group. The progressive increase in the probability of recurrence over two years was the greatest after discontinuation of lithium. For the patients who eventually discontinued lithium, the cumulative probability of recurrence in two years was 0.08 on lithium and 0.58 after stopping it. The probability of recurrence was unchanged over the duration of the study for patients who continued to take lithium. There was no evidence of a lithium withdrawal syndrome within three months of stopping the drug. The results support the view that the everyday clinical use of lithium as prophylaxis is beneficial in unipolar depression.

It is now widely accepted that lithium is effective in the treatment of acute manic states (Noyes, 1969; Davis & Farm, 1971; Lewis & Winokur, 1982) and also in the prophylaxis of bipolar illness (Schou & Baastrup, 1967; Zis & Goodwin, 1979; Prien *et al*, 1973, 1984). However, the indications for lithium in unipolar depression are less clear. An acute antidepressant effect when prescribed alone has been shown in some (Goodwin *et al*, 1969, 1972; Johnson, 1974; Worrall *et al*, 1979; Khan, 1981) but not all studies (Fieve *et al*, 1968; Stokes *et al*, 1971), and the prophylactic efficacy in unipolar depression has been the subject of considerable debate. A positive effect of lithium in reducing the frequency of relapse has been found in many uncontrolled (e.g. Baastrup & Schou, 1967; Grof *et al*, 1970; Kane *et al*, 1982) as well as controlled studies (Prien *et al* 1973; Coppen *et al*, 1976, 1978; Glen *et al*, 1984). However, other uncontrolled studies (Stancer *et al*, 1970; Bouman *et al*, 1986) have failed to find such an effect. The only prospective randomised trial of lithium prophylaxis in unipolar depression which failed to show an effect was that of Prien *et al* (1984). This study did discern a positive benefit in bipolars using the same design so that it was an important negative finding.

The present study has examined retrospectively the consequences of lithium discontinuation in a clinic population of unipolar depressed patients previously admitted to the Royal Edinburgh Hospital, the principal psychiatric teaching hospital serving the City of Edinburgh. The rate of recurrence of patients who discontinued lithium has been compared with that of a control group who continued to take it. The purposes of the study were twofold: (a) to assess the efficacy of lithium by comparing the outcome in these two groups, and (b) to examine the possibility of a lithium withdrawal syndrome occurring during the first three months of follow-up as occurs in bipolar patients (Mander, 1986).

Method

Any retrospective study is dependent on the quality of the case records. In the Royal Edinburgh Hospital, studies are facilitated by well assembled, typewritten clinical notes, and careful, consistent diagnosis using ICD-9 (World Health Organization, 1978). In addition, demographic details are recorded in a computerised database by the Lothian Psychiatric Case Register. The lithium office separately records biochemical data for all patients taking lithium, noting the serum levels and reminding doctors of the need for further checks. These databases enabled identification of all patients with a diagnosis of manic–depressive illness, depressed type (ICD-9 Code 296.1), who were discharged on lithium between 1 January 1970 and 31 December 1986.

Patients were included in the study if, at this index admission, they:

(a) satisfied DSM-III criteria for major depression (American Psychiatric Association, 1980)
(b) had remained symptom free for at least three months after discharge
(c) had no diagnosis other than unipolar depression before starting lithium
(d) were established at this index admission on lithium alone for prophylaxis of their recurrent unipolar illness.

These lithium-treated patients represented 158 out of a total of 2301 patients identified over the study interval with a diagnosis of unipolar depression. They were further divided into those who eventually discontinued lithium and those who continued to take it. Of the group who discontinued lithium, there were several further problems that resulted in the exclusion of 13 patients from the group before final analysis. These were:

(a) other psychotropic medication was being taken at the time of subsequent discontinuation of lithium
(b) it was not clear from their case notes or contact with the patient's general practitioner when lithium was discontinued
(c) they were clearly unwell at the time of lithium discontinuation.

Outcome was determined by time to readmission (again with unipolar depression). Since the patients remained resident in Edinburgh, they were unlikely to be admitted to any other centre, although this could not be determined with certainty. No particular bias should have been introduced by admissions lost in this way. Time to readmission was analysed using the Actuarial Life Table (Fleiss *et al*, 1976) in two phases. For patients who *discontinued* lithium (group A), the first phase was defined as the time from admission to the study (three months after index admission) to the time of lithium discontinuation (a mean of 29 months (range 4 months–11.5 years); the second phase was a two-year follow-up from the time when lithium was discontinued. Control patients who did not discontinue (group B) had the first phase defined from three months after the index admission, exactly as for patients who discontinued lithium, but with the end defined by matching these group B patients with patients in group A for duration of lithium treatment; again, phase II was a two-year follow-up. In this way, groups A and B could be compared for two phases: I, in which both were on lithium for a comparable time; and II, in which group A discontinued lithium and group B continued to take lithium, over two years.

Lithium levels were available from routine monitoring and provided a check on compliance, which appeared to be good. Most levels will have been determined 10–14 hours after the last lithium dose.

Cumulative probabilities from life-table analysis have been compared as a summary χ^2 and individual probabilities from a critical ratio derived from their standard errors as recommended by Fleiss *et al* (1976).

Clinical data have been compared for patient groups defined either by lithium discontinuation or by outcome (recurrence or remained well). Normally distributed parametric data have been compared using Student's *t*-test (unpaired, two-tailed); categorical data have been compared using χ^2 tests.

Results

The 145 patients whose fortunes form the basis of this study, were middle aged, predominantly female (104 women), and had, on average, at least three admissions to hospital with unipolar depression before starting lithium. The group who subsequently discontinued lithium were entirely comparable with those who did not. There were no statistically significant differences in sex ratio (10:30 v. 31:74, respectively), mean (s.d.) time since the first illness episode (7.7 (7.1) v. 6.5 (8.6) years), the time spent in hospital (16.4 (16.8) v. 15.4 (19.8) weeks), the number of admissions before introduction of lithium (3.7 (2.4) v. 3.3 (2.4), or their lithium levels after its introduction (0.66 (0.15) v. 0.72 (0.13) mmol/l). The only factor on which the groups differed was age at first episode: those who subsequently discontinued lithium were slightly younger (44.8 (14.9) v. 51.8 (15.3) years, $P<0.03$).

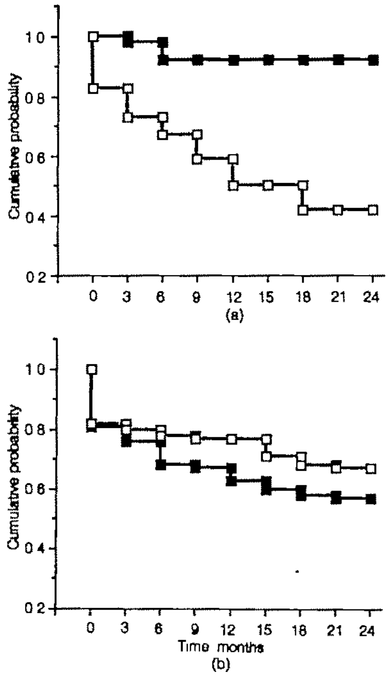

FIG. 1 Life-table presentation of the cumulative probability of recurrence in patients taking lithium. (a) Group A is shown after starting lithium (■—■) and after its discontinuation (□·······□). The difference between the two profiles was unlikely to have arisen by chance ($\chi^2=11.85$, $P<0.001$) (b) Group B is shown for two treatment phases in both of which the patients took lithium. Phase I (■—■) shows the probability of recurrence after starting lithium prophylaxis and is directly comparable with the phase of lithium treatment for group A; group A did better than group B during active treatment ($\chi^2=10.26$, $P<0.01$). Phase II (□·······□) shows the probability of recurrence in group B for a second period of continuing lithium treatment, arbitrarily defined by matching to the times of lithium discontinuation of patients in group A. The two phases of lithium prophylaxis are associated with a very similar probability of recurrence, but group B did significantly better than group A during phase II ($\chi^2=5.5$, $P<0.02$)

Life-table analysis revealed that patients who subsequently discontinued their lithium had been significantly more likely to stay well in the two years after starting it than those who continued it (Fig. 1). Because of this, the probability of being well after two years of lithium prophylaxis was significantly higher in the group who then discontinued lithium (0.92 v. 0.57, $z=5.20$, $P<0.01$). In other words, discontinuation was strongly associated with successful treatment.

The life-table analysis showed a significantly worsened outcome for group A, who discontinued lithium (Fig. 1), relative both to how they were before stopping lithium and to group B in either phase. The probability of remaining well for two years after stopping lithium was only 0.42. The risk of recurrence over the first three months after withdrawal (high for bipolar patients) is not significantly increased (Fig. 1).

Thus lithium withdrawal was the major factor determining the probability of recurrence. However, the clinical features of patients who were readmitted were also of interest. The 53 patients (15 men, 38 women) (21 discontinuing, 32 continuing lithium) readmitted in phase II were compared with the 50 (11 men, 39 women) (10 discontinuing, 40 continuing lithium) who remained well and were followed up throughout the 24-month follow-up interval. As would be expected, there were more cases admitted from the group who discontinued lithium ($\chi^2=3.8$, d.f. 1, $P<0.05$), but they did not differ on any of the other factors examined. Thus, mean (s.d.) for recurrent cases compared with that for those remaining well showed no difference in age at first depressive episode (49.1 (17.1) v. 50.3 (13.6), duration of history before prophylaxis (6.4 (7.2) v. 7.4 (9.0) years), total time in hospital before lithium (16.8 (19.0) v. 14.5 (19.1) weeks) or lithium level (0.70 (0.15) v. 0.71 (0.13) mmol/l).

Discussion

Because this study was retrospective, the patients were not randomly allocated to discontinuation or continuation of lithium. Nevertheless, the groups differed only on the age at the time of the first episode, which we believe is unlikely to have affected our overall results, since the seven-year difference is clinically unimportant. In any event, age was not significantly related to recurrence in this study, a finding that agrees with other work (Carroll, 1979).

It appears that those patients who stopped their lithium were those who were deriving the most benefit, which is consistent with our observations in clinical practice. Patients who do well and have been free of illness for an appreciable time are often those that request to discontinue the drug. Further, they may be more likely to do so when young. It is reasonable to assume that psychiatrists are also more likely to agree in these circumstances. It is unlikely that the group discontinuing lithium had less severe illnesses, since they had spent the same time in hospital as the other group before the introduction of lithium. Even if their illnesses had been less severe, this would result in a bias in phase II against the demonstration of a deleterious effect of lithium withdrawal.

The probability of remaining well over two years for those who continued taking lithium (57%) suggests a result similar to that found by Schou (1979), of a one-year probability of 78%, although Peselow *et al* (1982) found a two-year probability of only 37%. These figures illustrate the quite poor prognosis of many patients with recurrent depressive illness. Furthermore, these patients did not derive as much benefit from lithium treatment as did the patients who chose to discontinue treatment. While this appears paradoxical, it is a corollary of what was discussed above: those patients who are doing rather less well, and are slightly older, may be prevailed upon to continue the drug more readily. Nevertheless, their prognosis on continuing lithium was significantly better than that for patients on stopping lithium.

Figure 1 shows that there is no excess of recurrences in the first three months in phase II. There is, therefore, no evidence of admissions occurring purely as a result of lithium withdrawal and the situation is, therefore, different from that seen for bipolar patients. Bipolar patients showed an appreciably increased rate of readmission in the three months after lithium withdrawal in a retrospective study fully comparable with this one (Mander, 1986). Furthermore, the early appearance of manic symptoms after the withdrawal of lithium was confirmed in a prospective, double-blind, placebo-controlled design (Mander & Loudon, 1988). We cannot conclude that depressive symptoms do not appear after lithium withdrawal in unipolar patients, only that the recurrence of symptoms is not severe enough to cause admission, but this is still an important finding. It may explain some of the controversy in the literature regarding the lithium withdrawal syndrome, because in different studies different proportions of bipolar and unipolar patients have been used (e.g. Sashidharan & McGuire, 1983). The absence of an excess of withdrawal admissions also makes interpretation of the life-table data in discontinuation studies less ambiguous in unipolar than in bipolar populations.

Overall, our findings provide further evidence of a prophylactic effect in unipolar illness. Previous negative findings may have been due to patient selection in prospective studies (Prien *et al*, 1984) or smaller and hence less powerful retrospective analyses (e.g. Bouman *et al*, 1986). The main practical conclusion is that patients who are doing well on lithium should be encouraged to persevere with it.

Our definition of the end of the index episode and our use of readmission as a criterion for recurrence were somewhat arbitrary. We relied upon the conservative interpretation of case notes to determine whether patients had been symptom free for at least three months after discharge. This should have reduced the number of early readmissions that were likely to have been relapses of the index admission, but may have excluded some patients whose clinical response was poorly documented. This is unlikely to have invalidated our comparison between patient groups or our interpretation of the effects of lithium withdrawal. The choice of readmission as a criterion of recurrence was again conservative and selected in favour of severe illness. The decision to admit is also clearly influenced by the availability of social support and alternative community services but we believe that the relative homogeneity of the services provided in the area served by the Royal Edinburgh Hospital will have protected us from what might be described as artefacts of patient management. Thus, we would be most surprised if the rate of hospital admission within a given patient group was not directly related to the level of symptoms. An advantage of using readmission as a criterion is that it carries direct implications for the use of expensive in-patient resources. Finally, we were unable to find clinical predictors of the course of illness that could account for the differences between out patient groups: this is consistent with other studies addressing the question of lithium prophylaxis (e.g. Bouman *et al*, 1986).

The most important limitation of retrospective studies is that patients do not enter different treatment groups at random. Therefore, bias could be introduced which might account for the findings. Indeed, we suspect that bias was introduced in the present study, because the patients who discontinued lithium tended to be younger, and were doing better as a group than the control population. However, such patients might have been expected to do well after discontinuing lithium, whereas they actually did badly. In other words, the bias introduced by non-random discontinuation could have prevented our finding an effect of lithium prophylaxis but was unlikely to account for what we did observe. Randomised studies do not have these disadvantages but may, nevertheless, recruit unusual and hence unrepresentative samples of patients; moreover they are often expensive and difficult to organise.

The advantage of retrospective studies of this sort is that they describe what actually happens in delivery of care, and so are crucial in complementing the findings from randomised trials. If effects described in research trials fail to be translated into clinical practice, then it is important to determine why not. In a different sense, the success with which an effect can be translated from research to practice offers one of the more meaningful ways in which a quantitative audit of treatment efficacy could be devised. This study strongly suggests that lithium is of prophylactic benefit in unipolar depressive illness. It should strengthen our intention to treat recurrent depressive illness with lithium in our everyday clinical practice.

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Monoamines and Abnormal Behaviour

A Multi-Aminergic Perspective

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Classical nosology has been the cornerstone of biological psychiatric research; finding biological markers and eventually causes of disease entities has been the major goal. Another approach, designated as 'functional', is advanced here, attempting to correlate biological variables with psychological dysfunctions, the latter being considered to be the basic units of classification in psychopathology. Signs of diminished DA, 5-HT and NA metabolism, as have been found in psychiatric disorders, are not disorder-specific, but rather are related to psychopathological dimensions (hypoactivity/inertia, increased aggression/anxiety, and anhedonia) independent of the nosological framework in which these dysfunctions occur. Implications of the functional approach for psychiatry include a shift from nosological to functional application of psychotropic drugs. Functional psychopharmacology will be dysfunction-orientated and therefore geared towards utilising drug combinations. This prospect is hailed as progress, both practically and scientifically.

Functional psychopathology: a look backward

From the very beginning, biological psychiatry has orientated itself towards nosology, a way of looking at behavioural disorders as distinct, separable entities, each with their own symptoms, causation, outcome and treatment. Finding a marker and, eventually, the cause of, for instance, schizophrenia, depression or panic disorder has always been the major goal of biological psychiatric research. The introduction of DSM–III (American Psychiatric Association, 1980) powerfully reinforced this approach.

For many years now we have advocated and pursued another route, termed the functional approach to psychopathology (van Praag & Leijnse, 1965; van Praag *et al*, 1975, 1987*c*). Essentially, this approach has three tenets.

(a) The basic units of classification in psychopathology are not syndromes or nosological entities, but psychological dysfunctions, such as disturbances in perception, cognition, memory and information processing. *They* are the elementary constituents of psychiatric syndromes.

(b) Nosological classification of a given psychiatric disorder is the first step in diagnosis. The next step is the dissection of the syndrome into its component parts (i.e. the psychological dysfunctions and their subsequent measurement). Analogies of two-tier diagnosing are provided by medicine. Myocardial infarction, for example, is a nosological diagnosis which will be routinely complemented by an analysis of its functional sequelae, in terms of, for instance, heart beat frequency and rhythm, conductance and output. Treatment is predicated on the functional analysis, not on the nosological diagnosis.

(c) In biological psychiatric research, correlations between biological and psychological dysfunctions should be sought.

Functional psychopathology is dimensional in orientation, viewing a given psychiatric disorder as a conglomerate of psychological dysfunctions, most of them nosologically non-specific and occurring in different severities and in different combinations in the various psychiatric syndromes. The nosological approach is categorical, viewing psychiatric disorders as discrete entities, each with its own causation, symptoms and course.

The term 'functional' is not identical with 'symptomatological'. Psychiatric symptoms are the behavioural expression of a psychological dysfunction, not the dysfunction itself. A visual hallucination for example is a symptom, a particular perceptual disturbance the underlying dysfunction. Within the functional framework, the latter is the target of biological research.

The following exposition is meant to illustrate our belief that the functional approach is a productive one.

The role of dopamine in abnormal behaviour

In the late 1960s we reported the occurrence of dopamine (DA) disturbances in depression, most notably in vital depression (major depression melancholic type, called melancholic depression here) (van Praag & Korf, 1971*a*). In some patients, the probenecid-induced accumulation of the major DA metabolite homovanillic acid (HVA) in cerebrospinal fluid (CSF) was decreased, indicating a lowered DA metabolism in the central nervous system (CNS), particularly in the nigro-striatal DA system, the region that is predominantly represented by CSF HVA. Guided by Parkinson's disease with its severe disturbances in motor functioning and profound DA depletion, we searched for correlations between lowered CSF HVA and motor retardation. Comparing groups of depressed patients with and without pronounced motor retardation and lack of initiative, the HVA accumulation after probenecid was twice as low in the retarded patients as in the non-retarded depressives and in a control group. Similar findings were reported by other investigators (Papeshi & McClure, 1971; Banki, 1977*a*; Banki *et al*, 1981).

If lowered DA metabolism in the nigro-striatal system underlies retardation and inertia, increasing DA availability could be expected to exert an activating and energising effect. The drug *l*-dihydroxyphenylaline (*l*-DOPA) was chosen to boost DA metabolism, since it increases this substantially, while having little impact on noradrenaline (NA) metabolism. Moreover, the increase in DA metabolism persists over time (van Praag & Lemus, 1986).

No overall therapeutic effects were found when *l*-DOPA (average dose, 260 mg/day) in combination with a peripheral decarboxylase inhibitor was first studied in a group of melancholically depressed patients who were biochemically undifferentiated. Subsequently, we compared the effect of *l*-DOPA (290 mg/day) and a peripheral decarboxylase inhibitor in melancholic patients with lowered post-probenecid CSF HVA and in such patients with normal HVA response. Motor retardation and inertia were significantly more pronounced in the former group. *l*-DOPA improved motor functioning and level of initiative in the low-HVA group and normalised the HVA level. Mood and hedonic functioning were not significantly influenced. Anxiety levels rose slightly, but significantly. In the group with normal HVA response, *l*-DOPA failed to exhibit therapeutic effects (van Praag & Korf, 1975).

The latter experiment was repeated in a different group of patients using *l*-tyrosine instead of *l*-DOPA (van Praag, 1986*b*). In contrast to *l*-DOPA, *l*-tyrosine, the precursor of the catecholamines (CA) DA and NA, significantly increases CSF 3-methoxy-4-hydroxyphenylglycol (MHPG) in humans, indicating increased NA metabolism. CSF HVA, on the other hand, rises only slightly, suggesting that tyrosine's impact on DA metabolism (in the nigro-striatal system) is less pronounced than that of *l*-DOPA. Again, studying melancholic patients with and without lowered CSF HVA accumulation after probenecid, we observed no therapeutic effect of tyrosine on motor retardation and inertia in either group. Anhedonia improved, but anxiety worsened (see later), so that total ratings did not change significantly. Normalisation of CSF HVA did not occur.

Based on these findings we hypothesised that diminished DA metabolism in the nigro-striatal system underlies decreased motor activity and lowered level of initiative, irrespective of nosological diagnosis (van Praag *et al*, 1975). In accordance with this is Lindstrom's (1985) observation that low CSF HVA occurs in schizophrenics with pronounced lassitude and slowness of movement.

When attempting to link the DA system to the ability to carry out goal-directed behaviour, it is necessary to take into account that such behaviour consists of different components. First, an initial stimulus is needed. This can be an instinctual drive, such as hunger, thirst or sex, an emotion, such as anger or a cognitive set (e.g. the realisation of having to prepare for a test). Next, the goals to respond appropriately to the stimulus have to be selected (e.g. to find food to satisfy hunger; to display aggressive behaviour to reduce anger or to move to a library and study in the case of an upcoming test). Then, the behaviour to attain these goals has to be initiated and sustained. Finally, signals should be set in operation when the mission has been accomplished so that the behaviour can be terminated. The term 'drive' is used to indicate an initial stimulus of an instinctual nature. It is also used for the entire process of initiation and completion of goal-directed behaviour. For the sake of brevity we use the term in the latter sense in this paper.

The sequence leading to goal-directed behaviour can be disturbed at any junction. The human data suggest that DA is involved in the mobilisation, facilitation and sustenance of goal-directed behaviour (see Fig. 1). The large pool of animal data accord with this conclusion (Crow & Deakin, 1985; Freed & Yamamoto, 1985; Ashton, 1987).

The role of serotonin in abnormal behaviour

Whither '5-HT depression'

Disturbances in central serotonin (5-hydroxtryptamine; 5-HT) metabolism were first reported in individuals with depression. The lowered post-probenecid minus baseline levels of 5-hydroxyindole acetic acid (5-HIAA, the major degradation product of 5-HT) in the CSF of such individuals indicated diminished 5-HT metabolism in the CNS (van Praag *et al*, 1970).

Subsequently, disturbances in other systems indicative of central 5-HT-ergic functioning were observed. First, disturbances in platelet 5-HT, such as deviations in 5-HT content and uptake and in the density of imipramine receptors, were reported. Blood platelets have been considered as a model of central 5-HT-ergic nerve endings. Furthermore, disturbances in the ratio of the plasma concentration of tryptophan and the so-called competing amino acids were found. This ratio determines the influx of tryptophan into the CNS, and CNS availability of tryptophan, in its turn, is an important determinant of 5-HT synthesis rate. Moreover, several hormonal challenge tests indicated disturbances in 5-HT-ergic functioning. Finally, psychopharmacological experiments with indirect 5-HT agonists such as the 5-HT precursor 5-hydroxytryptophan (5-HTP), the selective 5-HT uptake inhibitors, and 5-HT antagonists, such as parachlorophenylalanine (PCPA), a 5-HT synthesis inhibitor, suggested that manipulation of central 5-HT can effect mood changes (Brown *et al*, 1990).

The data most directly related to central 5-HT metabolism are derived from CSF studies, although these studies are the most cumbersome to conduct. The inter-relation between the various 5-HT indicators is insufficiently known. (For review on 5-HT and depression see: van Praag, 1982; Goodwin & Post, 1983; Brown *et al*, 1990).

Initially, depressed patients with and without demonstrable disturbances in central 5-HT seemed indistinguishable in psychopathological terms. Therefore, in 1971, based on the data available at that time, the concept of biochemical heterogeneity of depression was introduced (van Praag & Korf, 1971*a*) which suggested that some forms of depression are linked to disturbances in 5-HT functions; others are not or are to a lesser extent only. The same syndrome, then, could be the ultimate outcome of various pathophysiological processes. As an analogy the anaemia syndrome was mentioned: anaemia patients present by and large the same symptoms, but the pathogenesis of the syndrome is heterogeneous, as is the treatment.

Subsequent studies, however, made the postulate of a separate category of '5-HT depressions' untenable (van Praag & Lemus, 1986). They indicated that increasing 5-HT availability *alone* is not a sufficient antidepressant measure. This argues against the concept of a depression type that is generated predominantly by central 5-HT disturbances, leading to decreased 5-HT availability.

Contra '5-HT depression'

In a double-blind, placebo-controlled, comparative study it was demonstrated that 5-HTP is an active antidepressant in patients admitted to hospital with rather severe forms of melancholic depression, while tryptophan is no better than placebo (van Praag, 1984). Both 5-HT precursors increase central 5-HT metabolism to an equal extent, as reflected in the probenecid-induced accumulation of 5-HIAA in CSF. They appeared to differ, however, in their effect on catecholamine (CA) metabolites in CSF (reflecting intracerebral metabolism of the mother amines). 5-HTP increases the metabolism of DA as well as of NA, while tryptophan does not (van Praag, 1983). In high doses (>5 g intravenously) trytophan even lowers DA and NA metabolism, possibly by interfering with the influx of tyrosine, the precursor substance of the CAs, in the CNS (van Praag *et al*, 1987*a*).

The ability of 5-HTP to stimulate CA metabolism is thought to be due to the presence of aromatic amino acid decarboxylase in CA-ergic nerve cells. Hence, 5-HTP is converted to 5-HT not only in 5-HT-ergic but also in CA-ergic neurons. In the latter, 5-HT functions as a false transmitter, as a consequence of which the synthesis rate of CA increases. The net functional effect of the two opposing processes (i.e. false transmitter formation leading to decreased function and increased CA production leading to augmented function) is probably heightened CA-ergic activity.

If 5-HTP's therapeutic superiority to tryptophan is related to its combined effects on 5-HT and CA systems, tryptophan's antidepressant efficacy would be expected to increase above the significance level by combining it with a compound capable of selectively increasing CA availability. In humans tyrosine increases CA metabolism, while 5-HT metabolism is not measurably changed. Combining tryptophan with tyrosine indeed led to significant antidepressant activity (van Praag, 1986*b*).

A second observation supports the hypothesis of 5-HTP deriving its therapeutic potential from its dual influence on 5-HT-ergic and CA-ergic systems. In about 25% of patients initially treated successfully with 5-HTP, the response subsided in the second month of treatment (van Praag, 1983). This phenomenon was paralleled by the normalisation of CA metabolism (again, as reflected in the CSF concentration of CA metabolites) while the metabolism of 5-HT remained increased. It was hypothesised that if clinical relapse and normalisation of CA metabolism were related, re-activating CA metabolism should restore clinical remission. This was indeed so: adding tyrosine to the 5-HTP regime in the group of relapsers once again led to subsidence of the depression and to a rise of CA metabolism (van Praag & Lemus, 1986).

These data suggest that *combined* augmentation of 5-HT and CA availability in the CNS provides the best conditions for antidepressant activity, and they argue against the existence of a separate diagnostic entity called '5-HT depression'. The behavioural effects of 5-HTP in monkeys have also been shown to be a function of its influence on 5-HT and CA systems (Raleigh, 1987).

If 5-HT disturbances are not linked to a particular syndromal subtype of depression, could they relate to particular psychopathological dimensions that may occur in depression, but might be lacking as well, explaining the occurrence of 5-HT disturbances in some, but not other, depressions (van Praag *et al*, 1987*c*)? This hypothesis seems plausible in the light of recent observations, the dimensions in question being disregulation of aggression and heightened anxiety. The relevant data are now briefly summarised.

5-HT and disturbed aggression regulation

In 1976 Asberg *et al* confirmed our findings of a subgroup of depressed individuals with low CSF 5-hydroxyindoleacetic acid (5-HIAA), and added that individuals who had attempted suicide accumulate in that subgroup. This finding was confirmed by many, although not all, investigators. Subsequent studies revealed moreover that low CSF 5-HIAA was not only observed in depressed individuals who attempted suicide but also in such individuals who were non-depressed and non-psychotic (e.g. personality disturbed) and in schizophrenic patients who had made suicide attempts because 'voices' had ordered them to do so, but who were not suffering from depression as defined by DSM-III (see van Praag, 1986*a*).

Lowered concentration of CSF 5-HIAA has also been reported in individuals with increased outward-directed aggression, but belonging to different diagnostic categories (Brown *et al*, 1979, 1982; Bioulac *et al*, 1980; Linnoila *et al*, 1983; Lidberg *et al*, 1984, 1985; Virkkunen *et al*, 1987). The biological study of aggression in humans is fraught with methodological difficulties such as the usually short duration of aggressive outbursts and the fact that these explosions are often associated with a host of social or toxicological (drugs, alcohol) factors which it is hard to control for. However, the fact that all seven studies published so far have reported low CSF 5-HIAA indicates that the finding is robust.

Dysregulation of aggression can be a major feature of depression, as evidenced by the greatly increased incidence of both suicidal behaviour and outward-directed aggression in depressed individuals (Plutchik *et al*, 1989). Depressed patients with low CSF 5-HIAA show not only an increased rate of suicide attempts but also an increased frequency of outward-directed aggression (van Praag *et al*, 1986).

The facts, then, seem to support the hypothesis that the signs of diminished 5-HT metabolism in the CNS, as originally observed in depression, are related to dysregulated aggression (see Fig. 1), irrespective of the direction it takes and of the nosological context in which it occurs.

5-HT and heightened anxiety

Anxiety is a second psychopathological dimension occurring in depression (although being by no means specific to that disorder) and several pieces of evidence indicate that it is related to 5-HT.

Therapeutic effects in panic disorders have been reported with 5-HT potentiating drugs, in particular 5-HTP, increasing 5-HT synthesis and the 5-HT uptake inhibitors such as clomipramine, trazadone, fluvoxamine and fluoxetine (Evans & Moore, 1981; Koczkas *et al*, 1981; Evans *et al*, 1986; Kahn *et al*, 1984, 1987; Den Boer *et al*, 1987). Even more interestingly, the effect of those indirect 5-HT agonists seems to be biphasic, at least in some patients (Kahn & Westenberg, 1985). An initial phase of increased anxiety, lasting 2–3 weeks, is followed by a phase of amelioration in which anxiety and panic attacks diminish. This biphasic effect was explained by postulating: (a) the hypersensitivity of post-synaptic 5-HT receptors; and (b) the relatedness of this phenomenon to the anxiety attacks. Under these circumstances, increasing 5-HT availability would first lead to additional stimulation of an already hyperactive 5-HT system and, hence, to clinical deterioration. Down-regulation of the receptor system due to increased 5-HT availability would eventually lead to amelioration of the anxiety disorder (Kahn *et al*, 1988*c*).

Recently, additional evidence favouring this hypothesis was acquired. Panic disorder patients were challenged with m-chlorophenylpiperazine (MCPP), a relatively selective 5-HT1 and 5-HT2 receptor agonist. Normals and patients suffering from major depression served as controls. The major findings were two-fold. Patients with panic disorder who were given MCPP showed a substantial increase in anxiety during the test, in comparison with those given placebo. In the control groups no significant response occurred, on either MCPP or placebo (Kahn *et al*, 1988*a*). Moreover, the cortisol response to MCPP was augmented in panic disorder patients; again, as compared with both the response to placebo and the MCPP response in the two control groups (Kahn *et al*, 1988*b*).

Another piece of confirmatory evidence is found in the development of drugs that diminish central 5-HT activity, and possess anxiolytic potential. Buspirone is an established anxiolytic. It has a high affinity for pre-synaptically located 5-HT1A receptors, and probably decreases 5-HT-ergic activity (Taylor *et al*, 1985). Ritanserin is a selective antagonist of post-synaptically located 5-HT2 receptors, for which anxiolytic actions have been reported (Ceulemans *et al*, 1985). Both drugs seem to be effective in generalised anxiety disorder; their effect in panic disorder has not yet been well studied. Recently, Jones *et al* (1988) reported animal data indicating that GR38032F, a selective antagonist of 5-HT3 receptors, appears to be a potential anxiolytic agent. In humans, the various 5-HT receptor systems cannot yet be studied separately.

If, indeed, in (certain) states of increased anxiety (certain) post-synaptic 5-HT receptors are hypersensitive, one would expect a compensatory diminution of central 5-HT synthesis, reflected by a drop of 5-HIAA concentration in CSF. No data are available on CSF 5-HIAA in anxiety disordered patients. In depressed patients, a negative correlation between CSF 5-HIAA and anxiety levels has indeed been observed (Banki, 1977*b*; Rydin *et al*, 1982).

The present data do not permit extending the 5-HT hypersensitivity hypothesis beyond panic disorder, but some preliminary data indicate 5-HT receptor hypersensitivity may be related to increased anxiety in general rather than to panic disorder alone. First, a correlation was found between cortisol response to MCPP and anxiety level as measured one week before the test in both panic disorder and major depression (Kahn *et al*, 1988*b*), suggesting a relationship between the general state of anxiety and 5-HT receptor hypersensitivity. Second, the relationship between CSF 5-HIAA and anxiety in depression, mentioned above, points in the same direction, and so does the finding that suppressors of 5-HT activity like ritanserin and buspirone are effective in generalised anxiety disorder.

A dimensional 5-HT hypothesis

Signs of decreased central 5-HT metabolism, then, seem to correlate not with a particular syndromal depression type but with particular affective disturbances – increased aggression and augmented anxiety (see Fig. 1). If these psychopathological dimensions are both related to disordered 5-HT metabolism, they would be expected to intercorrelate, and indeed this is so. In a recent series of studies on predictors of suicide and violence in groups of psychiatric patients with a range of diagnoses, we found trait anxiety to be strongly correlated with both suicide risk and violence risk (Apter *et al*, 1990).

The data favouring a dimensional 5-HT hypothesis, linking 5-HT disturbances to disordered regulation of aggression and anxiety across diagnoses, seem fairly convincing.

The role of noradrenaline in abnormal behaviour

Inside the pleasure principle

So far we have discussed MA-ergic involvement in the regulation of drive and affect. Both domains are frequently disturbed in depression (in particular in major depression, melancholic type) but such disturbances are by no means specific to that disorder.

A third major sphere of dysfunction in melancholic depression relates to the ability to experience pleasure. Normally much of what we do, or think, is not emotionally neutral but provided with a charge; globally speaking, a positive or a negative one. It generates pleasure or displeasure, in whatever way that may be defined. Anhedonia, the inability to experience pleasure, is a typical symptom of melancholic depression. It is one of the few almost pathognomonic symptoms in psychiatry.

Anhedonia is unlike flat affect or emotional blunting as may be observed in schizophrenia. Flat affect pertains to a global inability to experience, or at least to express, emotions; anhedonia indicates the inability to link a particular mental or motor activity with gratification, a quality that has been 'stamped in' when similar activities were carried out in the past. In melancholic depression, the emotional defect is 'localised' and there is no global defect in experiencing or expressing emotions. On the contrary, particularly negative emotions such as sadness, guilt, and shame are felt with profound intensity. In very severe cases of melancholic depression the ability to experience discomfort is also impaired. Mental and physical activities that usually would be experienced as aversive have less capacity to produce this effect.

Behaviour is strongly motivated by the attainment of pleasure and the avoidance of discomfort. The decreased ability to experience such modalities will reinforce the state of drive reduction, characteristic in melancholic depression.

In animals, brain systems have been found that code for rewarding and aversive experiences that occur naturally. The so-called reward and punishment systems (Olds & Milner, 1954) are, at least in part, MA-ergically innervated, the former by CA, the latter by 5-HT. DA is thought to be particularly involved in mobilising and orientating goal-directed behaviours. DA neurons constitute a 'go-system' or 'incentive-system' guiding the organism to areas in the environment associated with reward (Crow, 1973). In addition, DA neurons have been implicated as a substrate for intracranial self-stimulation (Wise, 1982; Mason, 1984). Although a role for NA in self-stimulation is less well supported than for DA, NA-ergic neurons seem to be active in 'emotional memory' (van Praag *et al*, 1988), the consolidation and retrieval of the emotional arousal induced by particular behaviours. Alternatively, NA-ergic tracts may constitute the connections between the neural representations of rewards anticipated from behaviours and the DA-ergic incentive system (Crow, 1973, 1977). NA-ergic mechanisms have also been implicated in selecting among a choice of possible behaviours to attain a particular goal, based on the experiences of reward or punishment that those behaviours elicited on prior occasions (Robbins *et al*, 1985; Ashton, 1987). A reward-related role of NA-ergic mechanisms in self-stimulation is supported by experiments with clonidine, an α_2-adrenergic agonist. This substance disrupts the rewarding component of brain stimulation in a selective manner, probably via activation of pre-synaptic α_2-receptors, and by doing so it curtails NA-ergic impulse flow (Franklin, 1978; Hunt *et al*, 1976). The catecholamine synthesis inhibitor α-methyl-*p*-tyrosine magnifies this effect (Hunt *et al*, 1976).

A noradrenaline hypothesis of anhedonia

NA-ergic mechanisms seem to enable the animal to couple the experience of reward or anticipated reward to a particular activity. The hedonic component of the melancholic syndrome can be conceived as a dysfunction of exactly that coupling mechanism. Situations that used to create pleasure are perceived as usual, but are now devoid of an emotional charge. Stein (1978) was the first to suggest depression to be "a disorder of positive reinforcement or reward function". We do not subscribe to this generalisation, but would suggest a more focused version of it. The hedonic disturbance in depression is, we submit, related to NA-ergic insufficiency, a transmission defect resulting in the inability to couple the reward component to mental or physical activities that used to carry such a charge (see Fig. 1).

The NA hypothesis of anhedonia fits into the classic information-processing theory of human cognition, in which two sets of structures are postulated, one for storing information, and the other for transferring it from one structure to another. In this model – and here we follow Kihlstrom's (1987) description – information from the environment is transduced into a pattern of neural impulses by the sensory receptors. This pattern is briefly held in the sensory registers, one for each modality, where it is then analysed by processes known as feature detection and pattern recognition. Information that has been identified as meaningful and relevant to current goals is then transferred to a structure known as primary or short-term memory. Here, the information is subject to further analysis, whereby the perceptual information is combined with information retrieved from secondary or long-term memory. In the primary memory, processes such as judgement, inference and problem solving take place. On the basis of an analysis of the meaning of the stimulus input, a response is generated. Finally, a trace of the event is permanently encoded in the secondary memory. It is in the short-term memory, one might assume, that the percepts (i.e. the perceptual information) is linked with the appropriate emotion (i.e. the emotion that was aroused in similar circumstances on previous occasions).

NA metabolism and NA-ergic functions have been studied in depression and in anxiety disorders. In the former, NA-ergic hypofunction has been postulated (Siever, 1988); in the latter, NA-ergic hyperfunction (Redmond & Huang, 1979). The concept of the NA-ergic nature of anhedonia, conceived as a psychopathological dimension, has not been systematically studied and, hence, is largely hypothetical. Some scattered data however, although tentative in nature, are sufficiently intriguing to prompt further investigation.

Preliminary studies of the noradrenaline hypothesis of anhedonia

Clozapine

Anhedonia can be pharmacologically induced by neuroleptics. With anticholinergic phenomena and extrapyramidal symptoms, it is one of the more common side-effects. The motor symptoms are thought to be related to nigro-striatal DA-ergic blockade. Most neuroleptics also block variably NA-ergic transmission. Conceivably, the latter effect could underlie the hedonic disturbances. Drugs that deplete NA stores such as reserpine, and drugs which reduce NA-ergic function via pre-synaptic inhibition, such as clonidine, can cause depression and anhedonia.

Clozapine is an 'atypical' neuroleptic, one of a group of compounds with more selective effects on meso-limbic and meso-cortical DA systems than their 'typical' counterparts. This could explain why their propensity to elicit motor side-effects is lower and their therapeutic potential is probably greater. In addition, clozapine has a profound blocking effect on NA receptors, particularly after acute administration (Burki *et al*, 1974; Bartholini *et al*, 1972; Buus Lassen, 1974).

The effects of clozapine (100 mg/day) administered for two weeks to six normal volunteers were studied in a placebo-controlled, double-blind, two-group design. Mood, drive and hedonic functions were assessed via a structured interview and recorded by a (blind) interviewer on a five-point scale. Effects occurred more or less sequentially. First, listlessness was reported (after 2–3 days); subsequently, there was lack of energy; and finally, in the second week, lowering of mood was noted (van Praag *et al*, 1990). The 'clozapine-syndrome' resembled that of mild vital depression (major depression, melancholic type). The hedonic decifit elicited by clozapine was significant and in the first few days was 'pure'; that is, not combined with other psychological disturbances.

Moreover, clozapine was compared with perphenazine (trilafon) in acutely psychotic patients without evidence of brain damage. Clozapine augmented the concentration of the NA metabolite 3-methoxy-4-hydroxyphenylglycol (MHPG) in CSF, while perphenazine did not (van Praag *et al*, 1976). The reverse proved to be true for the DA metabolite HVA; its concentration rose during perphenazine, but not after clozapine. These data suggest that clozapine, in contrast to perphenazine, has a blocking effect on post-synaptic NA receptors, conceivably accounting for the hedonic defects observed in the 'clozapine-syndrome'.

Tyrosine

In depressed patients, tyrosine, the precursor of DA and NA, increases the CSF concentration of HVA and MHPG (van Praag & Lemus, 1986), indicating that tyrosine furthers CA synthesis and metabolism. It has no overall therapeutic effect in depression, but is not devoid of pharmacological activity. In a preliminary experiment, discrete components of the depressive syndrome did change significantly. This was particularly true for anxiety which increased and for hedonic functioning, which improved (van Praag *et al*, 1990). These preliminary findings suggest that tyrosine exerts a measurable effect on NA availability while also significantly reducing anhedonia.

Clonidine

Clonidine, in low doses, stimulates pre-synaptic α_2-adrenergic receptors, leading to diminished NA release. Administered to normal individuals, it reduced paired-associate learning (Frith *et al*, 1985). Sedation is an unlikely explanation because clonidine did not affect other memory functions. It decreased blood pressure, suggesting diminished NA-ergic impulse flow, but this effect could of course be totally peripheral.

This finding suggests that NA neurons are involved in the coupling of the neural representation of associated stimuli. This is as far as one can go; it does not imply that these neurons are involved in linking an emotional memory trace ('pleasure') with a particular behaviour.

Hormonal studies

The growth-hormone response to clonidine (supposedly mediated by post-synaptic α_2-adrenergic receptors in the hypothalamus) has been shown to be blunted in endogenous depression compared with neurotic depression (Checkley *et al*, 1984). The most important distinction between the two syndromes with regard to symptoms is the presence or absence of anhedonia (van Praag 1962, 1989). Conceivably, the NA-ergic deficit and the hedonic disturbances correlate, but data to this effect are not available.

Another hormonal disturbance observed in major depression is blunting of the plasma cortisol response to desmethylimipramine (DMI), a tricyclic antidepressant and a selective inhibitor of NA uptake (Asnis *et al*, 1985). Correlating blunted DMI response with items of the SADS (Schedule for Affective Disorders and Schizophrenia; Endicott & Spitzer, 1978) related to anhedonia, a significant correlation was found with 'lack of reactivity', but not with 'loss of interest' or 'social withdrawal' (van Praag *et al*, 1990). The study was a retrospective one, and no specific attempts had been made to delineate the symptom of anhedonia from those that are somewhat similar but basically different.

A dimensional monoamine hypothesis

In this paper, we have discussed evidence of MA-ergic dysfunctions underlying the major domains of psychological dysfunctioning in melancholic depression: drive, affects and hedonic functioning. Sufficient evidence is available to implicate DA in drive reduction and 5-HT in disturbed aggression regulation and heightened anxiety. Tentatively, a link has been proposed between NA-ergic dysfunction and anhedonia (Fig. 1).

Both 5-HT-ergic and DA-ergic systems (Green & Deakin, 1980; Williams & Davis, 1983; Fuenmayor & Bermudez, 1985; Curzon *et al*, 1985) and 5-HT-ergic and NA-ergic systems (Ferron *et al*, 1982;

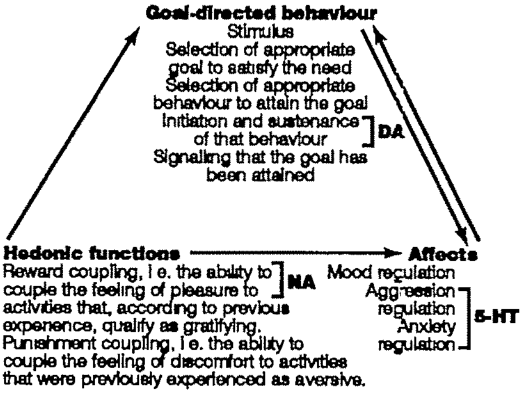
Goal-directed behaviour
Stimulus
Selection of appropriate goal to satisfy the need
Selection of appropriate behaviour to attain the goal
Initiation and sustenance of that behaviour
DA
Signaling that the goal has been attained
Hedonic functions
Reward coupling, i e. the ability to couple the feeling of pleasure to activities that, according to previous experience, qualify as gratifying.
NA
Punishment coupling, i e. the ability to couple the feeling of discomfort to activities that were previously experienced as aversive.
Affects
Mood regulation
Aggression regulation
Anxiety regulation
5-HT

FIG. 1 Summary of major domains of psychological dysfunction in melancholic depression, and hypothesised MA-ergic dysfunctions (NA = noradrenaline, DA = dopamine, 5-HT = 5-hydroxytryptamine) underlying these (e.g. 5-HT is implicated in disturbance of the affective components of anxiety and aggression).

Reinhard *et al*, 1983; Manier *et al*, 1984; Feuerstein & Hertting, 1986; Heal *et al*, 1986; Asakura *et al*, 1987; Devau *et al*, 1987) are strongly mutually intertwined. If the psychological dysfunctions under discussion are linked to MA-ergic dysfunctions, the psychological dysfunctions would be expected: (a) to often go in concert; and (b) to influence each other's severity. Disorders of drive, mood and hedonic functioning meet in the melancholic syndrome, but no systematic data are available about their quantitative interaction. Clinical experience, however, suggests that such interactions do occur. Anhedonia reinforces lack of drive and tends to aggravate mood lowering. Mood lowering in its turn has a negative impact on drive, while drive reduction itself probably acts as a mood-lowering force.

Disturbances in drive and in regulation of anxiety and aggression are by no means specific for melancholic depression, but also occur in other behavioural and in certain neurological disorders. This could explain the nosological non-specificity of MA disturbances. Signs of decreased DA metabolism have, for example, been found in retarded depression and in Parkinson's disease; 5-HT disturbances have been reported in depressed individuals who have attempted suicide but also in subjects with other diagnoses who have tried to kill themselves and in patients with outward-directed aggression irrespective of diagnosis. 5-HT disturbances have, moreover, been implicated in panic disorder, but the odds are that they relate rather to increased anxiety across diagnoses. Anhedonia, on the contrary, seems to be relatively characteristic for melancholic depression. The same would therefore be expected to be true for the alleged signs of NA-ergic hypofunction, but no data are as yet available.

So far, no data implicate particular MA-ergic disturbances in the mood component of melancholic depression. It is true that this issue has hardly been studied, but such a lack of data could also be explained by hypothesising mood disturbances to be secondary to other depressive dysfunctions, possibly lack of drive and anhedonia. A few observations support this. Katz *et al* (1987) demonstrated that the therapeutic effects of amitriptyline in depression are manifest already in the first week of treatment. The first symptoms to decrease are anxiety and hostility, and they do so in correlation with plasma amitriptyline levels. Mood improvement follows later in the second and third week (Katz *et al*, 1990). We found that in 100 patients admitted for first or second episodes of melancholic depression, anhedonia was the single most frequent initial symptom (42%), with drive reduction second most frequent (31%). Mood lowering was indicated as a first symptom by only 11% of the sample. Also, it should be noted that electroconvulsive therapy (ECT) works initially to increase motor behaviour and only subsequently to change mood (Fink, 1979).

Recently, Cloninger (1986, 1987) advocated the dimensional approach for the biological study of personality disorders. He introduced a system of personality variants based on three dimensions: novelty seeking, harm avoidance, and reward dependence – traits he additionally linked to particular MA-ergic systems. The scheme is elegant but lacks empirical foundation and to define and measure such distinctions in personality make-up will be very difficult. To view biological variables occurring in psychiatric disorders from the vantage point of psychopathological dimensions is feasible. Whether the same is true when personality dimensions are substituted for psychopathological dimensions remains to be seen. Considering the present state of diagnostic methodology, we predict that Cloninger went one step too far.

Functional psychopathology: a look forward

MA disturbances found in psychiatric disorders look chaotic if viewed from a nosological vantage point, being demonstrable in so many different diagnostic entities. Interpreting them from the functional viewpoint brings order to the apparent chaos. Thus, MA research clearly demonstrates the usefulness of the functional approach in psychopathology. The implications of this approach, however, reach beyond the boundaries of biological psychiatry in several important ways.

First, they reach into the realm of clinical psychopharmacology. Several psychotropic drugs have turned out to be diagnostically non-specific. Tricyclic antidepressants are efficacious in certain forms of depression *and* in certain anxiety states. Clomipramine is an effective antidepressant *and* a useful treatment in obsessive–compulsive disorder. Carbamazepine is an established antiepileptic agent *as well as* an asset in the prophylaxis of episodic mood disorders. These observations are puzzling from a nosological but not from a functional point of view. A particular drug could influence a particular functional system in the brain, and thus, a particular set of psychological dysfunctions, irrespective of diagnosis. The greater the biochemical specificity of a drug then, the greater the chance it will be nosologically non-specific, but effective against certain (clusters of) psychological dysfunctions, across diagnoses.

Once correlations between psychological and biological dysfunctions have been established, the next step will be the search for drugs that can selectively correct the biological dysfunctions. Such drugs would be expected to ameliorate the corresponding psychological dysfunctions, irrespective of nosological diagnosis. Within a nosological frame of reference treatment is directed towards a particular disorder, and within a functional framework towards the underlying or resulting dysfunctions. In the former approach treatment is preferably restricted to one drug; in the latter approach combinations of drugs will be used, contingent on the existing spectrum of dysfunctions. Functional psychopathology will lead to functional psychopharmacology and this will tend to use drugs in combination. Elsewhere, we have given an (hypothetical) example of transnosological indications of a group of psychotropic drugs – the selective 5-HT reuptake inhibitors, the first group that seems to possess a high degree of selectivity, at least as far as MAs are concerned (van Praag *et al*, 1987*b*). The objection could be raised that neuronal systems do not act in isolation, but interact with numerous others. That does not mean, however, that selectivity is illusory and that limiting a drug's primary actions is meaningless. Fluoxetine, for example, is a selective inhibitor of 5-HT uptake with little effect on NA reuptake. Its impact on NA-ergic functioning is not negligible, but certainly much less than that of a drug that primarily inhibits the uptake of both MAs, such as amitriptyline. Indeed, perfect selectivity is unattainable, but that goal can certainly be advanced.

Goal-directed, dysfunction-orientated polypharmacy is not a chimera, but a strategy legitimately employed in other medical disciplines. For example, once a myocardial infarction has been diagnosed and the cardiac condition functionally analysed, drugs will be administered to regulate the discrete disturbances in, for instance, rhythm, conduction, frequency and output. These drugs are often prescribed simultaneously. Thus, this represents goal-directed, dysfunction-orientated multipharmacy. It is in this direction that we expect to see clinical psychopharmacology develop. It is *the* scientific approach to drug treatment, at least as long as we cannot address the cause(s) of the disease directly.

A second field that will benefit from a functionally orientated psychopathology is that of measurement of abnormal human behaviour. So far, the presence of a psychiatric syndrome or symptom is expressed as a rough estimate; that is, as mildly, moderately or severely present, or anywhere in between. Many psychological functions and dysfunctions, however, are measurable in real quantitative terms and with considerable precision and reliability. Functional psychopathology will lay the groundwork for a scientifically based psychopathology.

Finally, the psychotherapies would benefit from the functional approach, because it would allow, much more precisely than is possible today, the delineation of mental domains that are dysfunctional from those that are still relatively normal. This would permit a rather precise definition of treatment goals and a more rational choice of treatment method. Treatment planning would thus be furthered and provided with a rationale.

Thus, functional psychopathology is not only a productive framework for biological research in psychiatry, but could, in addition, be evolving into a forceful catalyst for psychiatry's ongoing scientific orientation.

We introduced the functional/dimensional approach in psychopathology as a complement of and not as an alternative to the nosological/categorical approach. We still fully subscribe to this standpoint, as much as we do to the view that the exclusive adherence to nosology has not served biological psychiatry well. In fact, it has been a factor stunting its growth.

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Neurological Soft Signs in Schizophrenia

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A standardised clinical examination which focused on neurological soft signs (NSS) was used by a neurologist blind to diagnosis to assess the presence of neurological soft signs. A comparison of NSS score was made among three groups of subjects consisting of 58 DSM-III schizophrenics, 31 of their healthy first-degree relatives and 38 normal controls. The schizophrenic group had significantly higher NSS total scores than normal controls but did not differ significantly from their first-degree relatives' group. The relatives' group showed higher NSS total scores than did normal controls although to a lesser extent than schizophrenics.

In recent years psychiatry has focused its attention on neurodysfunctional correlates of schizophrenia and increasingly sophisticated neuroradiological, neurophysical and neuropsychological studies have been pursued for this purpose (for a review see Nasrallah & Weinberger, 1986).

Among various abnormalities, neurological signs, either 'hard' or 'soft', were reported by some studies to show a higher incidence in schizophrenics than normals (for a review see Heinrichs & Buchanan, 1988). 'Hard' signs have predictive localising power, usually referrable to specific lesions of nuclei, tracts, or nerves. 'Soft' signs represent abnormal performance on less specific tests, none of which by themselves indicate a clearly localisable central nervous system (CNS) lesion. Included under 'soft' signs are such phenomena as dysdiadochokinesia, astereognosis, mirror phenomena, choreiform movements, primitive reflexes, diminished dexterity, sensory extinction, and cortical sensory loss. In other studies, patients with schizophrenia have shown more neurological soft signs (NSS) than those with affective illness (Walker, 1981; Quitkin *et al*, 1976) but no more than those with a history of sociopathy (Quitkin *et al*, 1976). Affected monozygotic twins demonstrated more neurological impairment than their discordant siblings for schizophrenia (Mosher *et al*, 1971; Pollin *et al*, 1966). These abnormalities also correlated with lower birthweights and neonatal problems (Pollin *et al*, 1966).

Torrey (1980) further reported findings on lateralisation of neurological signs, supporting the hypothesis implicating left hemisphere dysfunction in schizophrenia. Kennard (1960) suggested that these signs were significant and probably related to dysfunction in subcortical integrating systems such as the basal ganglia or the brainstem. Moreover, several attempts were made to clarify a possible relationship between neurological signs and outcome (Kolakowska *et al*, 1985), intelligence quotient (Quitkin *et al*, 1976), medication, demographic variables, and localisation sites (Heinrichs & Buchanan, 1988), but there were no unambiguous conclusions.

Comprehensive studies report abnormalities on integrative sensory functions (Quitkin *et al*, 1976), motor co-ordination, and complex motor acts (Heinrichs & Buchanan, 1988); however it is not clear whether abnormalities in each of these functions occur together in a subgroup of neurologically impaired schizophrenics or whether these subgroups are characterised by impairment in diffuse systems (Heinrichs & Buchanan, 1988).

Interpretation of these results has been complicated by several factors. Firstly, the aetiology of the neurological abnormalities remains unclear. There is evidence of an association between increased neurological signs and a history of obstetric complications with schizophrenia (Campion & Tucker, 1973; McNeil & Kaij, 1978), but the causal links remain uncertain. Secondly there is a problem of specificity. A number of studies have looked for and found an excess of neurological abnormalities in other psychiatric patients, both children with behavioural disorders (Kennard, 1960; Woods & Eby, 1982) and adults with bipolar affective diseases (Quitkin *et al*, 1976; Nasrallah *et al*, 1983; Mukherjee *et al*, 1984).

Because of the renewed interest in this field, we designed a NSS study to explore the presence of neurological abnormalities in schizophrenics, their first-degree relatives and normal controls, using a standardised neurological examination.

Method

Fifty-eight schizophrenic patients (SCZ) (37 men, 21 women) were selected in an unbiased fashion from consecutive admissions to our university ward from the catchment area served by the hospital. They were admitted for clinical relapse and not for research purposes. Selection criteria for patients were: (1) DSM-III (American Psychiatric Association, 1980) diagnosis of chronic or subchronic schizophrenic disorder; (2) no history of neurological disorder, drug abuse or alcoholism; (3) no history of electroconvulsive therapy (ECT) and no significant side-effects from neuroleptic medication; (4) no history of institutionalisation, defined as a stay in hospital for more than six months; (5) ability to live in the community. Schizophrenic subjects had been ill for an average of 11.14 years (s.d. 8.51). Their ages ranged from 18 to 57 years (mean (s.d.) 34.82 (10.74)).

Thirty-one first-degree relatives (FDR) (18 men, 13 women) participated in the study: they were siblings ($n=28$) and parents ($n=3$). Their ages ranged from 18 to 59 years (mean (s.d.) 39.19 (11.11)). Only one relative of each patient was assessed for their psychiatric status. This was done by direct interview. Relatives with evidence of any DSM-III axis I or II disorders were excluded from the study. However, we did not find any DMS-III disorder in the 'index' relative interviewed and none of these subjects were receiving neuroleptic drugs. In selecting relatives for neurological examination, preference was given first to siblings and then to parents. For 27 patients no collaborative relatives were found.

Thirty-eight normal controls (NC) (22 men, 16 women) were screened for this study from family-practice clinics, by an experienced psychiatrist. Their ages ranged from 18 to 57 years (mean (s.d.) 36.15 (12.38)). All these subjects had no history of mental disorder in first-degree relatives.

Members of the NC group were matched according to age, sex, and educational level with the SCZ sample whenever possible.

Neurological examination

Based on previous work (Quitkin *et al*, 1976; Cox & Ludwig, 1979) we selected 26 items, seven of which were bilateral (i.e. left and right) and significantly related to 'soft signs' dysfunction. This allowed us to record the neurological examinations in a standardised format (see Appendix 1). All participants received this standard neurological examination administered by a staff psychiatrist (SdC) and a senior neurologist (VM) who were blind to research diagnoses.

The severity score was in all cases the sum of the scores of the 26 items assigned by the senior rater (VM); this was expressed as a NSS total score. For training purposes the two raters examined the subjects simultaneously and rated independently.

Reliability

To ensure inter-rater reliability, the two raters had evaluated simultaneously the neurological status of 30 mixed subjects independently from this study, and recorded the results separately. Inter-rater reliability was found to be 0.76 (Spearman correlation) for the total score. To test the reliability of observing each sign, a kappa analysis (coefficient of agreement) was done for those items scored 0 or 1, measuring them as a nominal scale. For the other items, scored as an ordinal scale, the Spearman correlation coefficient was used.

Statistical analysis

The NSS total scores were square-root transformed and analysed with an independent group one-way analysis of variance (ANOVA) and Scheffe's statistic (Nie *et al*, 1975). A parametric ANOVA instead of a non-parametric one-way ANOVA was used to achieve a better statistical control of the *a posteriori* comparisons. Age and educational level were statistically analysed by one-way ANOVA without transformation. Pearson's product-moment correlation coefficient was used where appropriate.

Results

Table I lists the demographic and clinical features of the three groups. An independent group analysis of variance (ANOVA) showed no significant differences in age and educational level between the groups.

One-way ANOVA demonstrated a significant difference in NSS total score between the three groups. Comparing the groups in pairs showed that both the SCZ and their FDR had higher total scores than the NC group, while no differences were found between SCZ and their FDR. The raw data are shown in Figure 1. A two-way ANOVA with status and sex as factors was performed in order to look

TABLE I

Demographic characteristics, clinical features and neurological soft signs (NSS) total score in the three clinical samples

	Schizophrenics *(n=58)* *mean (s.d.)*	*Normal controls* *(n=38)* *mean (s.d.)*	*First-degree relatives* *(n=31)* *mean (s.d.)*
Age	34.82 (10.74)	36.15 (12.38)	39.19 (11.11)
Educational level: years	8.00 (3.64)	8.52 (3.10)	6.90 (2.85)
Current drug dosage[1]	271.87 (215.52)	–	–
NSS total score[2]	12.63 (4.79)	4.07 (2.53)	9.80 (2.42)

1. Converted to clorpromazine milligram equivalents (CPZ mg/eq).
2. One-way ANOVA ($F(2;124)=44.59$, $P<0.0001$): schizophrenics vs controls, $P<0.0001$; controls vs first-degree relatives, $P<0.0001$; schizophrenics vs first-degree relatives, NS.

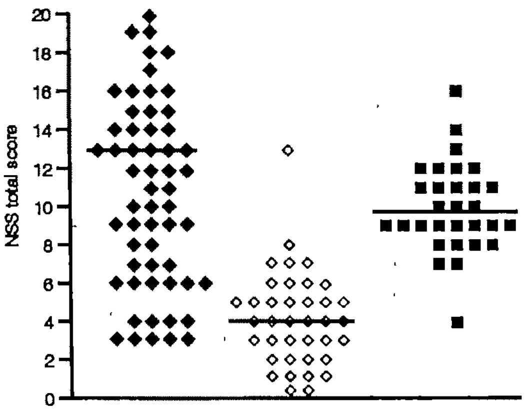

FIG. 1 Scatter diagram showing neurological soft signs total scores for each schizophrenic patient (◆), first-degree relative (■) and normal control (◇) (—— represents mean score for each group).

for a possible sex effect on NSS total score, but the finding was not statistically significant ($F(1;121)=0.006$,NS).

Age was significantly correlated with NSS total score among the NC group only ($r=0.42$; $P=0.009$). Educational level was significantly correlated with NSS total score among SCZ only ($r=0.55$; $P<0.0001$). To explore the age-at-onset effect on NSS, we split the sample of 58 patients into two groups, using the median of age at onset (24.5 years). Unpaired t-tests showed no significant differences between the early-onset (<24.5 years) and late-onset (>24.5 years) groups with regard to NSS total score.

Neither length of illness nor current neuroleptic drug dosage correlated with NSS total score. To discover possible effects of neuroleptic medication on each neurological sign scored as a nominal scale, we matched patients with zero scores with those who scored one, with current neuroleptic dosage as the dependent variable. A t-test for unpaired data did not show significant differences. A Spearman correlation coefficient was then calculated for the ordinal scored items. No significant correlations with current neuroleptic dosage were seen. Some coefficients were even negative (i.e. the higher the drug dosage, the lower the item score).

As to the inter-rater reliability of observing soft signs, kappa analysis yielded values ranging from 0.37 to 0.76. Indeed, these reliability coefficients were quite different but this was mainly due to the infrequent occurrence of some signs. In fact the kappa value is a function of the base rate of the measured event, as recently discussed by Spitznagel & Helzer (1985), so that less frequent signs yielded higher kappa values. For the other items, Spearman correlations ranged from 0.69 to 0.85.

Discussion

The results of this study support prior findings indicating a significant NSS increase in schizophrenics compared with control groups. Despite the increasing attention paid to the study of neurological abnormalities in schizophrenia (for a review see Cadet *et al*, 1986; Manschreck, 1986), surprisingly little attention has been paid to the development of a structured and reliable procedure for rating NSS.

Although the instrument we adopted is of demonstrated reliability, we are aware that there is a lack of validation for it. This might reflect the absence of an elaboration of the meaning of NSS in terms of neurofunctional localisation. Further studies examining the relationships between NSS and known risk factors (e.g. perinatal insult, family history, season of birth), clinical characteristics (e.g. age at onset, pre-morbid functioning, diagnostic subtypes, negative symptoms, cognitive impairment, treatment response, chronicity), and other neurodiagnostic procedures (computerised tomography, evoked potential, positron emission tomography, neuropsychological tests) may help to find external validators of the NSS construct. Subsequently, studies of internal validity and content validity might find factors associated with signs particularly useful in further research.

A second issue we explored concerns the presence of abnormal NSS in a substantial quota of a high-risk population (the patients' relatives). Our results indicate that schizophrenics and also their relatives showed more severe neurological soft signs than control subjects. This supports the view that these signs may be the result of a pathophysiological process that is probably family-related and confirms the results reported by Kinney *et al* (1986). Because of our lack of family-history data for the other first-degree relatives, we cannot control the information variance due to the illness heterogeneity in terms of genetic loading (De Lisi *et al*, 1987) and we are unable to make inferences about the inheritance of this trait.

Nonetheless, familial studies of high-risk groups for schizophrenia pointed out the presence of more neurological abnormalities in the offspring of schizophrenic parents than in matched control subjects (Marcus *et al*, 1985).

Statistical analysis of the data revealed no significant relationship between current neuroleptic drug dosage and presence of NSS in the schizophrenic patients. Despite these results, caution must be urged in not discounting potential interactional effects between medication and diagnostic groups on the relative incidence of soft signs. Nonetheless, the observation that the first-degree relatives' group, as a drug-naive population, shows a higher level of NSS than controls, seems to confirm that neuroleptic medication has minimal or no effect on NSS.

The strategy of testing first-degree relatives should be pursued as a matter of course. This strategy has been followed mainly for eye movement studies

(Holzman, 1987) and for psychophysiological studies (Friedman *et al*, 1982; Saitoh *et al*, 1984) with encouraging results, ruling out the possibility that these indices might represent trait-markers in schizophrenia (Erlenmeyer-Kimling & Cornblatt, 1987).

Recent views about thalamic dysfunction in schizophrenia ruled out the possible common role of the same thalamic regions in both neurological soft signs and smooth pursuit eye movement (SPEM) impairments (Oke & Adams, 1984). In a preliminary report Thaker *et al* (1988) found an association between NSS and SPEM impairment. Because of the reported SPEM and NSS impairment in first-degree relatives of schizophrenic patients (Holzman, 1987; Kinney *et al*, 1986) these signs appear to be strong candidates for biological markers of schizophrenia. Taken together, these observations suggest the need to study NSS further in the first-degree relatives of schizophrenics and in people considered to be at risk of the disorder to establish the potential usefulness of the NSS as a trait marker for schizophrenia.

Appendix –
Condensed neurological examination

Palmomental test The palm is stroked with a key and the chin is observed for the characteristic slight movement. A positive response is any reflex movement of the chin. Score 0–2 (0–1 left; 0–1 right).

Snout reflex The upper lip of the patient is percussed with a percussion hammer and the perioral region is observed for the characteristic movement. A positive response is any reflex movement of the region. Score 0–1.

Suck reflex This reflex is elicited by the examiner's finger which runs from the cheek to the mouth's omolateral commisure. A positive response is any sucking movement of the mouth. Score 0–2 (0–1 left; 0–1 right).

Blink reflex This reflex is elicited by gentle taps of the glabella repeated at one second intervals. An abnormal response is a failure to habituate the reflex after five taps. Score 0–1.

Gaze impersistence The patient is asked to gaze at a point for some seconds. Any gaze impersistence during 30 seconds is scored as abnormal. Score 0–1.

Ocular vergence The patient is asked to follow with the eyes a moving target along the visual axis. Any defect of the ocular vergence is scored as abnormal. Score 0–1.

Nystagmus The patients' eyes are observed for the presence of a nystagmus as he gazes at an object that moves around him describing an angle of 60 degrees respectively on the left and on the right-hand side. Score 0–2; (0–1 left; 0–1 right).

Oral apraxia Difficulty in the patient's ability to show his/her teeth and puff out cheeks is scored as abnormal. Score 0–2; (0–1 for each sign).

Motor perseveration in spoken commands test This test attempts to reveal motor perserveration of the patient in spite of the verbal request to perform a simple act. For example, the examiner asks the patient to "point to your nose with a finger". Any perseveration of the asked act is scored as abnormal. Score 0–1.

Grasp reflex Each palm is stroked by the examiner with moderate pressure from the wrists to the fingers. A positive response is any grasping motor movement of the hand and is graded on a scale of 0 to 5. Score 0–10; (0–5 left; 0–5 right).

Blunt vs sharp discrimination test The patient is touched in a random but standardised fashion with the sharp and blunt ends of a pin on the dorsal surface of the left and right hands while the eyes are closed. The patient says "sharp" or "blunt" upon stimulation. Any misperception on the left and on the right side is scored independently on a scale of 0 to 5. Score 0–10; (0–5 left; 0–5 right).

Graphestesia The patient is asked to identify a number that the examiner traces on the back of the patient's wrist, on the left and the right side alternately. The patient is informed that the number will be from zero to five. Three stimuli are applied in a random but standardised fashion. Any misperception on the left and on the right side is scored independently on a scale of 0 to 3. Score 0–6; (0–3 left; 0–3 right).

Simultaneous bilateral tactile extinction The patient is seated, with the right hand, dorsal surface exposed, on the right thigh and the left hand, dorsal surface exposed, on the left thigh, with eyes closed. The patient is informed that the examiner will touch him/her in one or more places, and that he/she should report exactly where he/she feels this. The following order was followed: right hand; right side of face; right side of face and right hand; left side of face and left hand; both hands; both sides of the face; right side of face and left hand; and left side of face and right hand. A positive response is an incorrect perception. A scale of 0 to 6 corresponds to the number of misperceptions. Score 0–6.

Complex motor acts The examiner watches the patient tie a shoe and scores the effort as positive if abnormal difficulty is exhibited on a scale of 1 to 5. A score of 1 corresponds to minimal difficulty and 5 to complete inability to perform the act successfully. No difficulty in performing the item is scored as 0. Score 0–5.

Imaginary acts The imaginary act of pouring and stirring coffee is observed objectively for omission of the essential parts of the act; holding a coffee pot, then pouring the coffee in a cup, replacing the coffee pot, taking the teaspoon, and stirring the coffee. Any major omission constitutes a positive score. A scale of 0 to 5 is used which indicates the number of omissions. Score 0–5.

Astereognosis One at a time a coin, key, and pen are placed into the patient's palm by the examiner. The patient must identify the object without vision and using only the hand, left or right respectively, in which the object has been placed. A positive response is a failure to identify an object. A scale of 1 to 3 indicates the number of failures for the right and for the left one respectively. Score 0–6 (0–3 left; 0–3 right).

Two-object test The patient is asked to perform four manoeuvres of increasing complexity with a pencil and a key, for example, "point to the pencil with the tip of the key". The score is based on a failure to complete any of the manoeuvres. Score 0–4.

Tapping-rhythm test The examiner taps out three different rhythms individually and the patient is asked to duplicate each one. A positive response is an erroneous response to any rhythm. Score 0–3.

Dysdiadochokinesia The patient's ability to perform rapid alternating movements is evaluated. The patient is asked, for example, to slap the palm and then the back of the hand rapidly and alternately on his own knee. A positive response is any pattern characterised by irregular rhythm, uneven force and breakdown of the alternating movements. It is graded on a scale of 1 to 2. Score 0–2.

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A Neuroendocrine View of ECT

STEPHEN J. COOPER, ALLAN I. F. SCOTT and LAWRENCE J. WHALLEY

> "A neuroendocrine hypothesis to explain the clinical activity of convulsive therapy is described. The hypothesis is based on the diabetes/insulin model and suggests that hypothalamic dysfunction with an insufficiency of a mood-maintaining peptide is the basis for affective disorders. Repeated seizures enhance the production and release of the hypothalamic peptide *antidepressin* – the active principle that relieves both neuroendocrine and behavioral abnormalities."

The summary quoted above is from an article by Fink & Nemeroff (1989). The present authors were invited to comment upon the study.

Stephen J. Cooper

Electroconvulsive therapy (ECT) remains an important although decreasingly used treatment for severe depressive illness, mania, and schizophrenia. Its clinical use is now largely for drug-resistant cases, and this is perhaps unfortunate from an academic view, as it may mean that much of the research carried out is on somewhat atypical cases. Nevertheless, ECT is an important form of treatment to study. Unlike psychotropic drugs, it is applied principally to the brain and its measurable biochemical effects are much more likely to have a central origin than are many of the effects we observe with drugs, which are often due to peripheral changes that are not necessarily mirrored in the brain. It is important, therefore, to try to discover the relevant therapeutic changes produced by ECT, as these may provide important clues to the fundamental biochemical disturbances which occur in disorders such as endogenous depression.

Interest in the possible involvement of neuropeptides in psychiatric illness has increased in recent years and detailed hypotheses have been developed (e.g. Charlton & Ferrier, 1989). While various alterations in peptide concentrations have been demonstrated in both animals and man with ECT, no clear relationship with therapeutic response has yet been identified for any. In this sense, the broad hypothesis advanced by Fink & Nemeroff is no better and no worse than the many hypotheses advanced for monoamine and other neurotransmitters. They criticise theories relating to other neurotransmitters by suggesting that the changes in these may be epiphenomena of the seizure activity. However, as yet, many of these have been inadequately investigated in the clinical situation, which these authors accept as being the most important one in which to judge the relevance of alterations in function of particular neurotransmitter systems. Furthermore, when we do not actually know which of the electrical/electrophysiological effects of ECT are important in the response to it, it may be premature to write off apparent biochemical epiphenomena. The authors suggest thyrotrophin-releasing hormone (TRH) as a possible candidate peptide, but some aspects of alterations of thyroid control also appear to be seizure epiphenomena. Scott *et al* (1989) have demonstrated this for changes in thyroid-stimulating hormone (TSH) following ECT.

In supporting the role of their putative hypothalamic peptide 'antidepressin', Fink & Nemeroff rightly point to the importance of vegetative symptoms in depressive illness. However, other symptoms of mood, motor, and cognitive disturbances, which involve centres outside the hypothalamus, are also important. While some brain peptides are widespread, not all are. On the other hand, monamine neurotransmitters are known to be widespread, and their pathways arise from fairly discrete nuclei, so that dysfunction in one of these systems may have a wide effect. Furthermore, not all depressives demonstrate all or even typical vegetative features, while schizophrenic patients may have none of them and yet respond to ECT. This suggests that we might have to invoke more than one important pharmacological effect if antidepressin relates especially to vegetative/hypothalamic changes.

The neurophysiological evidence argues for an effect of ECT on centrencephalic brain structures which, according to the thesis, include structures such as the hypothalamus that are important in the regulation of release of many peptides. However, such structures also include the nuclei for monoamine systems such as the locus coeruleus (noradrenaline), raphae nuclei (serotonin) and substantia nigra/ventral tegmental area (dopamine).

Nevertheless, this seems an interesting and tenable hypothesis, although clinical evidence in favour of it still seems rather non-specific, its principal clinical support perhaps coming from failure to substantiate a clear role for other neurotransmitters in ECT response. The possible link between ECT and alterations in corticotrophin-releasing hormone (CRH) may be the most fruitful avenue for research. Charlton & Ferrier (1989) have proposed a useful hypothesis relating hypothalamic–pituitary–adrenal function, and hence also CRH function, to depression. It is important, however, that there are various neurotransmitter influences on the secretion of CRH (Gillies & Grossman, 1985). Also, CRH itself may modify neurotransmitter function, such as that of noradrenergic systems (Valentine *et al*, 1983). Thus, all arguments for or against peptides versus neurotransmitters may be circular in the end. Indeed, my resolve after reading this paper is that we should be attempting to study aspects of peptide and neurostransmitter function in tandem, since interactive alterations in both may result in separate clinical effects.

Allan I. F. Scott

The great attraction of the neuroendocrine hypothesis is its simplicity. This is more obvious when this paper is compared with other papers in the same special edition of *Convulsive Therapy* (No. 3, vol. 5, 1989) on the mode of action of ECT. Hypotheses based on cerebral hemispheric asymmetry, 3H-DADLE binding, the c-*fos* proto-oncogene and the epileptic Mongolian gerbil are all included. The neuroendocrine hypothesis may be simpler, but, as Oscar Wilde said, the truth is rarely pure and never simple. Oscar may not have known how to behave in public, but I think he predicted something important about the mode of action of ECT.

The hypothesis in question can be considered in two stages. Firstly, that affective disorders are the result of a lack of a mood-regulating peptide ('antidepressin') and that ECT works by stimulating its production. In support of the hypothesis, Fink & Nemeroff remind us that the mode of action of ECT may be distinct from the presumed mode of action of other antidepressant treatments. They draw attention to the growing awareness that several peptides which were at first described only in relation to their role in the regulation of endocrine function are widely distributed through the central nervous system and may have other biological effects relevant to cognition and behaviour. Adrenocorticotrophic hormone (ACTH), TRH, CRH, vasopressin, and the encephalins are cited as examples.

Exciting as these ideas are, even the authors accept that if 'antidepressin' exists, its identity is unknown. Nevertheless, the neuroendocrine hypothesis is proposed as a model for the study of the mode of action of ECT. I have several reservations about this proposal.

Affective disorders are conceptualised as a simple increase or decrease in the function of one regulatory system, but contemporary models suggest they result from a failure of regulation in one or more systems, rather than simply too much or too little activity in any one system (Siever & Davis, 1985). In other words, the proposed model fails to take account of interactions between different neurotransmitter systems in the regulation of mood and behaviour.

Moreover, it seems premature to focus on one single biological effect of ECT in attempts to explain its mode of action. A stated conclusion of the neuroendocrine hypothesis is that changes in activity of other neurotransmitters such as serotonin, noradrenalin, and dopamine are epiphenomena of seizure activity and are unrelated to the clinical efficacy of ECT. However, I shall hang on to my shares in the catecholamine hypothesis and await a better takeover bid.

The second stage of the hypothesis states that the abnormalities in pituitary–adrenal and pituitary–thyroid activity are reversed when patients recover from their mood disorder, and that antidepressin is responsible for both processes. The basic premise that neuroendocrine abnormalities which are common in depressed patients are reversed when patients recover is not supported, though, by the results of recent studies. The most widely researched neuroendocrine abnormality is the failure to suppress the release of cortisol by the administration of dexamethasone, i.e. non-suppression in the dexamethasone suppression test (DST). It is now clear that recovery from depressive illness after ECT can occur independently of normalisation of the DST. The majority of persistent DST non-suppressors improve markedly or remit (Lipman *et al*, 1986; Fink *et al*, 1987). Moreover, the use of serial DSTs has shown that clinical improvement during a course of ECT is compatible with a change from suppression to non-suppression (Coryell, 1986). The finding that recovery from depressive illness can occur independently of normalisation of abnormalities in hypothalamic function has been confirmed with regard to the TRH stimulation test (Decina *et al*, 1987) and shortened rapid eye movement sleep latency (Coffey *et al*, 1988). Of course, in some individuals, neuroendocrine dysfunction may be a marker of a biological vulnerability to affective disorders rather than an associated feature of the presence of a mood disorder. Nevertheless, these observations suggest that it is highly unlikely that one regulatory system alone, let alone a postulated mood-regulating peptide, is responsible for both mood and neuroendocrine abnormalities.

These criticisms are not meant to imply that brain peptides cannot be involved in the regulation of mood, nor that peptides released during ECT have no role in the biological effects of the treatment. Brain peptides may modify signal transduction in monoamine neurotransmitter pathways which regulate mood. There is now good evidence that in the rat, an opioid peptide released into the cerebrospinal fluid contributes to the observed anticonvulsant effect of ECT. However, an epileptic Mongolian gerbil and antidepressin still have one feature in common. I would not recognise either of them.

Lawrence J. Whalley

Neuropeptides are present in the brain at concentrations much below those of 'classic' neurotransmitters. Outside the hypothalamic–pituitary system (where neuropeptides have an obvious endocrine function) their precise role remains unknown. Co-localisation of neuropeptides with established neurotransmitters has suggested a possible 'neuromodulatory' action at these sites. The hypothesis that neuropeptides are involved in the pathogenesis of mental illness, particularly depressive disorders, remains largely untested, not through lack of interest but because of major methodological problems encountered in clinical research on the behavioural actions of endogenous neuropeptides. It is the purpose of the Fink & Nemeroff paper to encourage psychiatrists to keep in mind a possible role of one or more neuropeptides among the causes of depression and the mode of action of ECT.

The idea that neuroendocrinology (specifically the study of neural control of endocrine functions) should be especially focused on ECT is not new. For at least 30 years, psychiatrists have postulated that the 'neurovegetative' symptoms of depression may have a common origin in limbic–hypothalamic dysfunction, and that neuroregulatory factors (many of which have been identified as peptides with well established behavioural actions in addition to their endocrine roles) present at these sites may be causally important in depressive illness (Carpenter & Gruen, 1978).

Certainly, this thought was in the minds of the Edinburgh group, led by George Fink, when in 1980 he initiated a series of studies that set out to describe systematically the endocrine events induced by ECT and to determine their relationship to improvements in specific symptoms. Some years earlier, the Oxford group, led by Grahame-Smith, had begun to examine the effects on serotonergic receptor function of repeated electroconvulsive shocks in animals. At about this time, a consensus view had emerged in British psychiatry that, contrary to popular opinion, there was a sound scientific basis for the efficacy of ECT, although not for its mode of action. Neuroendocrine studies in Edinburgh established for the first time that ECT had quite selective effects on hormone release by the pituitary gland. Against expectations of a massive, non-specific release of available hormones, a discrete pattern of secretion was observed that was remarkably consistent within individuals during early treatments.

Attempts to link these clinical studies with the promising animal work reported by Grahame-Smith *et al* were beset with difficulty, not least because of lack of information on the role of serotonergic neurones in neuroendocrine systems but, of particular relevance to the Fink & Nemeroff paper, because of major methodological difficulties concerning the detection and measurement of central neuropeptide release. One approach, which eventually proved uninformative, was to devise a sort of neuroendocrine algebra in which each of the neuropeptides known to be involved in the control of hormones released after ECT was considered and then, by exclusion, the minimum number of identified neuropeptides that could be involved in the hormone effects of ECT was deduced. In the face of a very considerable amount of data, no convincing role for any single neuropeptide emerged. How then should the Fink & Nemeroff paper be reconsidered? Specifically, does their speculation about neuropeptides and the mode of action of ECT facilitate progress towards a new testable hypothesis?

Central to their argument is a role for a novel neuropeptide that they term 'antidepressin'. In support of such a role, they cite several strands of evidence that include reports of abnormalities in endocrine function found in depression. However, these studies have not remained unchallenged and when proper account is taken of the effects of diet, weight loss, sleep disturbance, exposure to and withdrawal from medication, intercurrent illness, age, sex and reproductive status, and the choice of inappropriate control subjects, then the residual evidence is not compelling. It is certainly not possible to state with conviction that there are established endocrine abnormalities in depressive disorders.

Like many before them, especially those who have examined the amine hypothesis of affective illness, Fink & Nemeroff consider that the latency of onset of action of ECT provides an important clue to the nature of those biological changes necessary for its antidepressant efficacy. While such a view was once commonplace, it was never supported by extensive evidence. In fact, when Crow & Johnstone (1986) reviewed "the gradual onset of action of ECT", they could identify only two studies that supported this belief, neither of which had been designed to test the latency hypothesis. More recently, the NIMH Collaborative Program on the psychobiology of depression (Katz *et al*, 1987) has re-examined this question and found that oral antidepressants act in those who respond within the first week of treatment. Likewise, Post *et al* (1987) have found 50% improvement among responders in the first week of treatment with ECT. It is by no means established that "latency of onset of action" is a consistent feature of ECT, and it certainly should not form an essential part of any hypothesis concerning its likely mode of action.

Fink & Nemeroff offer little practical guidance on the detection and measurement of their proposed 'antidepressin', and basic scientists interested in the neurobiology of ECT are unlikely to hurry to prepare research programmes designed to determine its role. They are much more likely to follow well trodden paths and continue to examine systematically the effects of ECT on aminergic transmission. Eventually, such studies may identify one or more neuropeptides that are released during seizure activity and capable of modifying central aminergic function. This laborious and painstaking approach, while less exciting than the potential discovery of a single abnormality that determines all the biological changes in depressive illness as suggested by Fink & Nemeroff, is more likely to unravel the complex series of neural events that follow ECT and their relevance to its antidepressant actions.

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The Systematic Collection of Patient Data in a Centre for Child and Adolescent Psychiatry

PHILIP D. A. TREFFERS, ARNOLD W. GOEDHART, JAN W. WALTZ and ELS KOUDIJS

Computerisation of case records has been slow to take place in child psychiatry, partly because of the amount of detailed and sometimes complex information required. A program for storing case records has been developed and it has proved of great value in epidemiological work, for example, regarding patient age structures and family compositions.

In adult psychiatry, work is being done to improve the organisation of case records and the elicitation of patient information (Lieff, 1987). In child and adolescent psychiatry the standardisation of case records and their computerised processing are as yet being exploited in only a limited way, because much elementary information about children and adolescents (e.g. composition of the family) is complicated. Moreover, it is necessary to include a large quantity of detailed data on development and on the child's parents.

Some initiatives have been developed (Thorley, 1982; Looney *et al*, 1984; Wong, 1987). The American Academy of Child and Adolescent Psychiatry has a Database Committee which has started developing a standard case record, presently being used in approximately 20 centres.

This article reports on the development of standardised, computerised case records, in a large centre for child and adolescent psychiatry. Its purpose is to improve clinical work and to create better conditions for scientific research.

In many institutions case records are often incomplete, even with respect to such things as demographic data. At times there are so many lacunae in the collected information that adequate patient care cannot be provided, and scientific research is limited. This is true even of the simplest forms of research, which are aimed at gaining insight into elementary data (referral age, classification, and length of treatment) of patients in an institution. A databank makes possible the determination of, for example, any shifts occurring in the patient population (e.g. Roghmann *et al*, 1982), and allows proper evaluation and follow-up work.

Development of computerised case records

The core information

In child psychiatric research widely diverse information is collected from various informants. The selection of data is largely determined by the questions which one seeks to answer with the help of the databank. Various possibilities have been described in the literature: the information collected might serve as a basis for policy decisions (Snyderman *et al*, 1979), or for diagnosis and classification (McDermott & Hale, 1982). One can also designate as core information the data necessary for selecting different forms of treatment, where minimum criteria are stipulated on which the choice for a certain treatment is to be based (Cohen, 1979). No reliable system for this approach has, to our knowledge, been developed for child and adolescent psychiatry. Nor is it likely to be developed in the future, as, in general, the decision algorithms cannot be unambiguously formulated.

Given the amount of data available, it is necessary to record only a selection of the data, such as the living situation of the child and the background and development of the problem.

Standardisation of the case records

A study group in the Academic Centre for Child and Adolescent Psychiatry Curium (the Netherlands) has been working on the development of a standard case record, to be used in both the out-patient clinic and the residential clinic. Approximately 200 children and adolescents are examined at the out-patient clinic each year, a number of whom are also treated. Fifty-four in-patients are also treated, during an average stay of 20 months.

We assume that the collection of information can offer material for research, in particular hypothesis-generating research, into a child and adolescent psychiatric population. In addition, the computerisation of standard case records offers the possibility of producing standard reports on individual children.

The standard case record is only a part of the complete patient records: there is a written component which includes, for example, the qualitative information and the impressions of the clinician.

The bases for developing the standard case record were:

(a) that only information would be collected as is, in principle, also collected for the 'routine' child and adolescent psychiatric examination

(b) that the databank must be complete, i.e. that it must be possible to obtain standard information in *every* case

(c) that the demands made on the time of the child, the family and the staff can be reduced by structuring the interview, which also ensures that important points are explicitly covered
(d) that the emotional burden for the child and the family must not be allowed to become too great.

The databank is *not* intended for clinical decision making based on computer-processed information.

With regard to the use of the information, regulations were drawn up which were submitted to the Medical Ethics Committee of the Medical School of Leyden University for review.

The selection of the data

The information gathered for the standard case record falls into four categories:

(a) demographic data and psychiatric history
(b) elementary anamnestic, test and diagnostic information (including history of pregnancy and parturition, functional development, diagnosis)
(c) functioning of child and family
(d) other (temporary) information, in conjunction with specific research projects.

Staff attitudes

There were, on the one hand, problems of a 'technical' nature which inevitably accompany greatly increased use of the computer. On the other hand there were doubts about the relevance of the standardised databank and about the value of the standard report generated by the computer, and these doubts led to incomplete or inaccurate information being recorded. The method used by psychiatrists with a psychodynamic orientation in particular entails collecting only that information which seems relevant for one specific child or family, and on the basis of associative considerations, intuition and tentative suppositions.

However, the introduction of the standard report was on the whole assessed positively by the staff, and it conferred reliability and accessibility to the information.

A number of members of staff viewed the introduction of computerisation as an impossible or senseless form of "application of a high technology tool to a low technology specialty" (Looney *et al*, 1984).

The description of family composition

Developing a form on which the basic information about the composition of the patient's family could be entered proved more difficult than was expected. The forms used for this purpose at various academic departments for child and adolescent psychiatry in the Netherlands proved insufficient; one drawback, among others, was that they could not accommodate the numerous forms of non-traditional family structures. In keeping with the approach used by Kellam *et al* (1977), we chose a method of describing the family structure in terms of the relation between the child and each adult present. Kellam *et al* found ten different types of relation, although not all ten proved useful for Dutch households (it is, for example, very unusual for relatives like a grandmother or an aunt to be part of the family household). Another problem is that Kellam *et al* used 'stepmother' and 'stepfather' as the only differentiations from natural parents. We use the terms 'biological', 'adoptive', 'foster' and 'step' to describe biological or adoptive parents and their partners living in the family home. If a child resides elsewhere (e.g. with relatives or in an institution) information is collected about the nature and degree of responsibility for the child assumed by the parent figures concerned. In this case we differentiate on the basis of the responsibility of the parent figure rather than the child's current place of residence.

Parents who bear primary responsibility for the welfare of the child are designated as parents providing primary care. The parent figures, or the institution, who, with the consent of the parents providing care, also care for the child, are termed "parent figures providing secondary care". This distinction is important if a child resides in an institution or with another family. In addition to the items mentioned above, information is also collected on:

(a) the nature of the changes which took place in the composition of the family (e.g. divorce or death of one of the parents)
(b) the time that the change took place
(c) the frequency of any contact between the child and the parent figure who is no longer part of the family household.

Another complicated matter is that regarding the 'types' of siblings a child can have. There are 26 possibilities where the sibling can be:

(1) a biological child, (2) an adopted child, or (3) a foster child of the two parent figures providing the primary care for the enrolled patient
(4) a biological child, (5) an adopted child, or (6) a foster child of the mother figure providing primary care for the enrolled patient
(7) a biological child, (8) an adopted child, or (9) a foster child of the father figure providing primary care for the patient
(10) a biological child, (11) an adopted child, or (12) a foster child of the mother figure providing secondary care for the patient
(13) a biological child, (14) an adopted child, or (15) a foster child of the father figure providing secondary care for the patient
(16) a biological child, (17) an adopted child, or (18) a foster child of the biological parent(s) no longer providing care for the patient
(19) a biological child, (20) an adopted child, or (21) a foster child of the adoptive parent(s) no longer providing care for the patient
(22) a biological child, (23) an adopted child, or (24) a foster child of the foster parents) no longer providing care for the patient
(25) a biological child of the step-parent no longer providing care for the patient
(26) a biological child of the co-parent no longer providing care for the patient

These are not 'academic' possibilities, but are drawn from practical experience.

The demographic data now being collected, using specially drawn-up forms, on the parent(s), the patient, and the parents no longer providing care (if applicable) include: age, education, occupation, job situation/source of income, date of marriage/living together/divorce/death, nationality and ethnological particulars. (A copy of this set of forms and the instructions are available from the first author on written request.)

The standard case record includes a number of items of information about the family in which the child lives. In addition to the relation between the child and the adults present, it also specifies the nature of changes which may have taken place in the composition of the family and the age of the child at the time of the changes. These data are incorporated into a summary description in the computer report. For scientific research it is also desirable to summarise this information in descriptions which are clearly related to practice and free of unnecessary detail. Kellam *et al* (1977) have investigated the construction of descriptive indices in the context of epidemiological research, but their approach has received little following among child psychiatrists. A well differentiated registration system allows comparison of family structures.

An illustration of use: the description of family composition

The data on the 267 children examined in the Curium outpatient clinic for child and adolescent psychiatry during January 1987 to July 1988 were used to produce summary descriptions of family composition. We describe below the correlation between these indices and the sex and the age of the child, as well as (when applicable) the relation to the length of the time between the child's loss of a parent and his/her referral. What must be emphasised is that the information is offered as an illustration; it is not our intention to present an exhaustive treatment of the subject.

The following two classifications of family composition were initially chosen for use in the study. Classification A is the simplest description, based only on the number of parent figures present (providing primary care):

A-I two-parent families; 199 (74%) of the children come from this type of family
A-II one-parent families, which count for 53 (20%)
A-III institutions, which provide primary care for 15 (6%) of the children in the group studied.

Classification B is according to type of parent figure. Distinctions are limited to those between biological, adoptive and foster/host parents in the following categories:

B-I families with two biological parents (152 children; 57%)
B-II two-parent families with one biological parent (27; 10%)
B-III families with two adoptive parents (11; 4%)
B-IV other two-parent families: foster-families, host families, relatives (9; 3%)
B-V one-parent families with a biological parent (50; 19%)
B-VI other one-parent families (3; 1%)
B-VII institutions (15; 6%).

Further differentiation of these classifications can be made in the case of the one-parent families (category A-II, or B-V and B-VI) and two-parent families with one biological parent (category B-II) by taking into consideration the cause of the loss of a (biological) parent, namely divorce or death. It is known that the effects of losing a parent through divorce differ in a number of respects from the effects of losing a parent through death. Extensive research has been done on how both divorce or the death of a parent affect the development of children (e.g. Van Eerdewegh *et al*, 1985). The sex of the parent providing primary care seems to be of less importance (Steinhausen *et al*, 1987). By taking into account the cause of the loss of the parent figure, we arrive at the following subcategories in classification B:

B-II-A two-parent families with one biological parent after the death of a biological parent (2 children; 1%)
B-II-B two-parent families with one biological parent after the divorce of the biological parents (25; 9%)
B-V-A one-parent families with a biological parent after the death of a biological parent (3; 1%)
B-V-B one-parent families with a biological parent after the divorce of the biological parents (42; 16%)
B-V-C one-parent families with an unmarried single mother (5; 2%).

However, such differentiation leads to small numbers in the subcategories.

Another differentiation can be obtained by including in the description the age of the child at the time he or she lost a parent. In this case a distinction is made between a loss before or after the age of six. The information was not available for five children who were examined only very briefly by a screening consultant. Adding these data to classification B yields the following subcategories:

B-II-A two-parent families with one biological parent, and the child *younger* than six at the time he/she lost a parent through divorce or death (17 children; 6%)
B-II-B two-parent families with one biological parent, and the child *older* than six at the time he/she lost a parent through divorce or death (9; 3%)
B-V-A one-parent families with a biological parent, and the child *younger* than six at the time he/she lost a parent through divorce or death (23; 9%)
B-V-B one-parent families with a biological parent, and the child *older* than six at the time he/she lost a parent through divorce or death (18; 7%)
B-V-C one parent families with an unmarried single mother (5; 2%).

Results

The ratio of boys and girls was nearly the same in the first two categories of classification A (70% v. 30%), but differed in the case of children referred by an institution (33% boys and 67% girls).

In classification B, of the patients who came from a family with two biological parents, 72% were boys and 28% were girls. The greatest deviations from this relation were found not only among children referred by an institution but also among those in the care of foster-parents, host families or relatives: 44% boys and 56% girls. From families with adoptive parents relatively more boys were referred: 82% boys and 18% girls.

Of the children from a family with two biological parents, 20% were younger than six years old, 55% between 6 and 12, and 25% older than 12. The distribution is similar for the children of adoptive parents, but those from other kinds of families were as a rule older. Of the children from institutions, 55% were older than 12, while 50% of the children in the care of foster-parents or host families were above the age of 12. In the remaining categories nearly 40% of the children were older than 12 at the time of referral.

The difference between the various categories of classification B with regard to both boy : girl ratios and age at referral constitutes an argument for using this more detailed classification for further research.

When the categories B-II and B-V were split into the subcategories based on age, as above, there appeared to be almost no differences in the boy : girl ratio and only a slight difference with regard to age at referral between the subgroups (children from families with one biological parent who lost a parent before their sixth year were slightly younger). The distribution was clearly varied, however, in the case of the interval between the time of the loss and referral. The intervening period was as a rule shortest for children from one-parent families who lost a parent after they were six years old: 11 of the 18 children were referred within two years of the loss, four after two to four years. The spread was greatest among children from single-parent families who lost a parent before their sixth year: five of the 23 children were referred within two years, six after two to four years, six after four to six years, and six after six years. Children from two-parent families with one biological parent were in general referred later: eight of the 26 children were enrolled four to six years after the loss, 14 children after six or more years. In this group there was nearly no difference between children who lost a parent before and after the age of six with regard to the interval preceding referral.

Discussion

Although definitive conclusions cannot yet be drawn, these results indicate that the number of parents, the kind of parenthood and (when applicable) the age at which a child lost a parent, should all be included in a summary description.

Research publications in child psychiatry have paid virtually no attention to such family relations; information of this sort is often not even included in the description of the research group. However, this information is indispensable. Nearly all decisions made in child and adolescent psychiatry take into account the specific details of family composition. In view of the relatively large number of patients from 'incomplete' families it is necessary to make these notions explicit and to test their validity. This is particularly true for epidemiological research, research into diagnostic categories, and the relation between diagnosis and treatment. From epidemiological research in the Netherlands it appears that approximately 7% of patients come from one-parent families (Verhulst *et al*, 1985). Of the children referred to Curium, more than 20% are from one-parent families. The ratio of boys to girls up to the age of 10 is approximately 3:1; after that the ratio quickly moves in the direction of 1:1. No clear explanation has been given for this change. Our data indicate that this change in sex ratio is largely dependent on the composition of the child's family. For children from families with two biological parents, the ratio of boys to girls up to the age of ten is 2.8:1, for older children it is 2.5:1. A greater shift takes place in the other groups. If all the children who have lost a parent through divorce or death are taken together, the ratio of boys to girls is 2:1 up to the age of ten, and 1.4:1 after that. If we limit ourselves to patients from families with two biological parents and from 'incomplete' families, it appears that up to the age of ten, 72% of the girls come from 'complete' families and that after the age of ten this figure drops to just 50%. The corresponding figures for boys are 78% and 67%. The relative increase in the number of girls with psychiatric disorders in early adolescence could be partly explained by the fact that the emotional problems of girls from divorced families make their appearance relatively later than those of boys (Kalter *et al*, 1985). This explanation presupposes that among children above the age of ten, girls with psychiatric problems are more often from 'incomplete' families than boys. Our data support this supposition. There is, however, no epidemiological information on this point. Psychiatric problems in children arise relatively often under the influence of environmental factors (probably more often than in adults). For example, there is the clinical impression that depression occurs relatively often among children from incomplete families.

Findings of this kind are also significant for scientific research into diagnostic categories in child and adolescent psychiatry, such as examining symptoms in terms of the internal construct validity of diagnostic categories for various groups of children. Consideration should also be given to a critical assessment of the relation between diagnosis and treatment indications. One of the most important variables in this connection is the composition of the family.

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Acute Blocking of Naloxone-Precipitated Opiate Withdrawal Symptoms by Methohexitone

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In a double-blind placebo-controlled trial of 18 patients, methohexitone blocked objective signs of opiate withdrawal caused by a bolus injection of naloxone. Furthermore, in continuing the naloxone therapy for 48 hours, no withdrawal signs appeared. Levels of withdrawal distress returned to normal levels within six days. This approach can be regarded as an effective and well tolerated withdrawal therapy with low drop-out rates.

Since Wilker *et al* (1953) demonstrated that an opiate antagonist can precipitate an abstinence syndrome in opiate-dependent subjects, opiate antagonists have been tried as therapeutic agents (Resnick *et al*, 1977; Charney *et al*, 1986) in opiate detoxification treatment. Recently Hendrie (1985) showed that high doses of naloxone are able to attenuate the withdrawal syndrome in opiate-addicted rats. Although Vlissides *et al* (1988) could not reproduce this study in humans because of the acute withdrawal syndrome precipitated by naloxone within minutes after administration, our earlier results confirmed Hendrie's data (Loimer *et al*, 1988).

Eisenberg (1985), finding that opiate-addicted rats pre-treated with phenobarbital have a more attenuated withdrawal response to naloxone than those pre-treated with other substances, suggested that, in humans, the acute onset of withdrawal symptoms after naloxone injection could be overcome by co-medication with a barbiturate. The aim of the following study was to test this assumption that the acute naloxone action in opiate addicts will be influenced by a barbiturate.

Method

Eighteen patients (three women, 15 men, aged 20–35 years), admitted to the Psychiatric University Hospital of Vienna for a short in-patient opiate detoxification programme, were studied. All patients satisfied DSM-III-R diagnostic criteria for opiate dependence (American Psychiatric Association, 1987) and generally had an opiate addiction history of 2–18 years. All patients abusing drugs other than opiates were excluded: to identify these, urine specimens were tested for opiates and other drugs of abuse by EMIT-tests before admission. All patients gave written informed consent to the study which previously had been approved by the ethical committee of the University of Vienna. The study was performed according to the principles of the Declaration of Helsinki. All patients taking part in this study were admitted to the hospital 24 hours before the beginning of the treatment and underwent a physical examination.

During the seven-day study, withdrawal symptoms were rated in two ways. Firstly, daily at 8 a.m., the patients completed a self-rating questionnaire based on that by Kolb & Himmelsbach (1938) which included 20 items associated with withdrawal distress: concentration, appetite, craving, pessimism, activity, anxiety, sleep, restlessness, mood, irritability, pain, sexual appetite, sweating, diarrhoea, insomnia, tremor, thinking about drugs, weakness, flushes, and aching bones and muscles. Secondly, during the double-blind trial and daily at 8 a.m., patients were rated by an independent psychiatrist on a nine-item scale, based on that by Wang (1974), which measured the presence and severity of withdrawal. Withdrawal symptoms were given different weight: yawning, epiphora, sweating, shivering (one point each); stomach pain, nausea, goose pimples, trembling, muscular pain (three points each); restlessness, vomiting (five points each). Scores were added to give a measure of withdrawal severity.

The patients were randomly allocated into a naloxone and placebo group (nine patients in each group) according to a double-blind design.

For the first two days of the study each patient received a single oral dose of morphine daily (100–300 mg morphine sulphate), the last being administered 12 hours before the

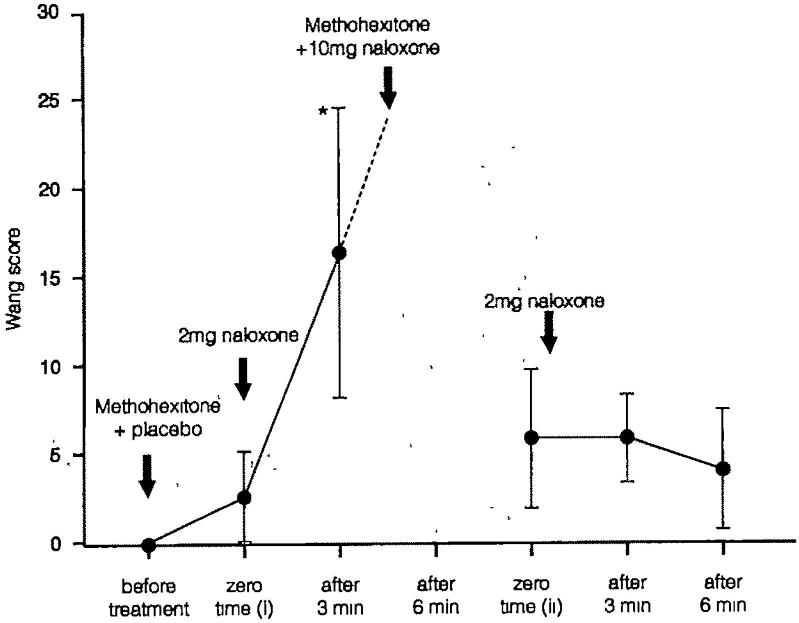

FIG. 1 Group B: effects on mean Wang score (s.d. shown by vertical lines) of a provocation test with 2 mg naloxone after placebo-methohexitone treatment and after subsequent naloxone (10 mg)-methohexitone treatment in opiate addicts ($n=7$; $^*P<0.01$; zero time = time after action of methohexitone (I) or methohexitone-naloxone (II) had worn off).

double-blind trial began. This ensured that no patient showed any withdrawal signs at the start. Twelve hours after this final morphine intake anaesthesia was induced in each patient by injection of 100 mg methohexitone (Brietal) without any pre-medication. Then the patients were intubated and artificially ventilated, and anaesthesia was maintained by a further injection of 400 mg methohexitone. Immediately afterwards 10 mg naloxone (Narcanti) (group A) or placebo (group B) was administered intravenously as bolus. When the narcotic effect of methohexitone had fully worn off (approximately 30–40 minutes later) patients were rated for withdrawal signs by an independent rater by means of the revised nine-item Wang scale. Thereafter, 2 mg naloxone was administered intravenously as a provocation test, and the Wang rating procedure was repeated three and six minutes later. In the event of severe withdrawal signs following the provocation test, indicating that the patient had received the placebo, brief anaesthesia was repeated by the injection of 250 mg methohexitone in total. Identical to the procedure in group A, patients then received 10 mg naloxone by means of a bolus injection. After the narcotic effect had again fully worn off, the whole rating procedure and the provocation test was done as before.

After this, the study was continued in an open design for both groups. All patients received intravenous naloxone (0.8 mg/h), for the following 48 hours. Observation of the patients then continued until the end of the study.

In order to observe all patients fully, admission time was generally seven days in total. During this time, urine samples were collected every day and tested for opiates, and as noted before, the self-rating and Wang scales were filled out every morning at 8 a.m.

Results

In group B (the placebo group), after the action of methohexitone had worn off, only minimal withdrawal signs were seen. However, a provocation test (2 mg intravenous naloxone) precipitated acute and severe withdrawal symptoms (Fig. 1). Because withdrawal signs in these patients were generally so severe, further rating was discontinued. Patients were anaesthetised again with methohexitone, which immediately interrupted all withdrawal symptoms.

In group A (the naloxone group), neither during barbiturate action nor after the anaesthetic effect had worn off were any but minimal withdrawal signs recorded, although they had received 10 mg of naloxone during the anaesthesia. Even the provocation test with 2 mg naloxone did not precipitate a change in the revised Wang score. Similar results were observed for group B following the second barbiturate application: after the injection of 10 mg naloxone, neither during nor after the methohexitone action were any severe withdrawal signs observed (Fig. 1). As with group A, a subsequent provocation test with 2 mg naloxone did not lead to changes in the Wang scores.

Following this treatment, when both groups received naloxone (0.3 mg/h) for a further 48 hours, no increase in withdrawal symptoms was observable in the Wang scale at any time.

In the self-rating tests, an increase in subjective

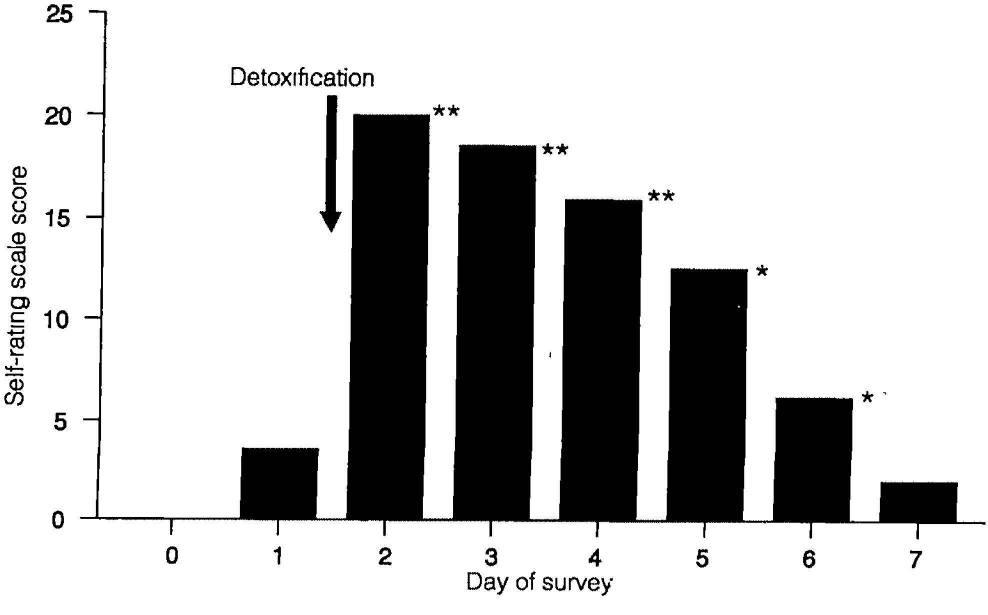

FIG 2 Mean score on self-rating withdrawal scale for all patients ($n = 18$) during seven days of study including period of detoxification treatment (*$P<0.05$, **$P<0.01$).

withdrawal distress was observed after the beginning of the described withdrawal regimen on the second day (Fig. 2). It steadily decreased during the next four days, even during the continued naloxone regimen, and base levels, similar to those which patients had shown during chronic opiate abuse, were seen after five to seven days. Furthermore, 48–72 hours after the start of the detoxification, all patients showed opiate negative urines.

This treatment regimen was well tolerated by the patients, none of whom dropped out of the study. All of the patients were discharged from the hospital after seven days of admission with only minimal levels of observable subjective or objective physical withdrawal symptoms. However, in our experience, patients do not require any special care after the acute detoxification procedure, and, in a routine schedule, might be discharged after two to four days.

Discussion

This study shows that the *acute* onset of withdrawal symptoms induced by naloxone in opiate addicts is blocked by the acute action of the barbiturate methohexitone. After administration of a high dose of naloxone, a dramatic provocation of withdrawal would be expected (Wikler *et al*, 1953). This was not observed when naloxone had been given during the pharmacological action of the barbiturate (group A).

In group B (placebo), shortly after the anaesthesia wore off, an acute withdrawal was provoked by only 2 mg naloxone. These results suggest that the severity of morphine withdrawal precipitated by naloxone decreases under the acute action of the barbiturate methohexitone, but cannot be prevented by a barbiturate *pre*-treatment. These data also indicate a paradoxical pharmacological action of naloxone: high doses of naloxone administered during a short barbiturate anaesthesia will prevent the onset of withdrawal signs. In group A, no distressing reaction was seen following a provocation test with 2 mg naloxone after the barbiturate action had worn off. These data confirm the observations of Hendrie (1985) in animals and our own results in humans (Loimer *et al*, 1988) that high doses of naloxone seem to suppress effectively the abstinence syndrome in opiate addicts. Although this paradoxical pharmacological action of naloxone cannot be explained by its known action on the opiate system, it seems that higher doses of naloxone are necessary as Vlissides *et al* (1988) have already suggested.

These observations suggest the basis for a fast and efficient detoxification treatment. In contrast to that of Gossop *et al* (1987) our study showed that patients with a long history of opiate addiction were symptom free five to seven days after detoxification. Unlike Brewer *et al* (1988), who described a naltrexone–clonidine detoxification treatment causing substantial discomfort for patients, we found that by continued naloxone infusion after high-dose naloxone loading during brief methohexitone anaesthesia, only mild objective withdrawal signs were observed. As Gold (1979) pointed out, many treatments causing abstinence syndromes lead to an early relapse. Our design offers a way to decrease such relapse during opiate detoxification: the fact that only mild (objective) withdrawal symptoms result make the treatment attractive to drug addicts afraid of such symptoms, while eliminating the need for heavy sedation for several days (Kleber & Riordan, 1982).

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To Weigh or Not To Weigh?

Frequency of Weighing and Rate of Weight Gain in Patients with Anorexia Nervosa

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The present study compares the rate of weight gain during refeeding in 15 anorectic patients who were weighed daily with that of 15 who were weighed three times per week. There was no significant difference between the two groups.

There is now a general consensus among clinicians that weight restoration must occur if psychological treatment is to be effective in the management of patients with anorexia nervosa. Behaviour-modification techniques have become an integral component of most refeeding programmes as they are easily mastered by paramedical staff and have been shown to facilitate weight gain (Garfinkel & Garner, 1982; Garner, 1985; Touyz *et al*, 1984, 1987).

Informational feedback has been reported to be an important component for successful treatment (Garfinkel & Garner, 1982; Wilson *et al*, 1985). Because of this, some have recommended that patients be weighed each morning during refeeding (Maxman *et al*, 1974; Agras & Werne, 1978) whereas others have warned of the iatrogenic dangers of intensifying the patient's preoccupation with body weight (Pardee, 1941). To circumvent this, it has been suggested that patients be weighed less frequently (Day, 1974) or that the frequency of weighing be negotiated with individual patients (Garfinkel & Garner, 1982).

To our knowledge, there has been no systematic study as yet which has compared the rate of weight gain during refeeding in patients who are weighed daily with those who are weighed less frequently while participating in a hospital programme under the care of the same consultant.

Method

Our sample consisted of 30 in-patients with anorexia nervosa, as defined by DSM-III-R (American Psychiatric Association, 1987). They were consecutive admissions to a specialised eating-disorders programme at a large teaching hospital and were all under the consultant care of one of the authors. The demographic and clinical characteristics of the patients are presented in Table I. They were divided into two cohorts. The first 15 patients were weighed daily whereas the next 15 patients were only weighed three times per week.

Patients were refed using a lenient flexible behavioural programme (Touyz *et al*, 1987). Such a programme is seen as more acceptable by the majority of patients, requires less nursing time, and is more economical as it allows more time for staff to engage in individual and group psychotherapy. In the present study, patients were told that they had to gain 1.5 kg a week at the time of their admission to the unit. Provided they complied with this requirement, they were free to move around the unit or attend outings to a nearby shopping complex. Furthermore, they were permitted to leave the unit on weekends for a few hours with relatives or friends. They understood that if they failed to achieve the weekly target of weight gain they would be

TABLE I

Demographic and clinical characteristics of the 30 anorexia nervosa patients (means and standard deviations)

Demographic and clinical characteristics	*Frequency of weighing* Daily	Three times a week
Age: years	20.13 ± 3.89	17.53 ± 2.7
Admission body mass index	13.77 ± 1.48	14.88 ± 1.42
Weight gain during refeeding: kg	9.59 ± 5.48	8.61 ± 3.5
No. of days of refeeding	56.33 ± 20.09	50.9 ± 15.37
Daily weight gain: kg	0.19 ± 0.12	0.17 ± 0.06

All differences between groups non-significant.

restricted to the unit until they had done so. For example, if patients only gained 1 kg in a week, they were restricted to the unit until they had gained the remaining 0.5 kg. However, if patients failed to make the weekly target of 1.5 kg over two consecutive weeks, they were restricted to the unit for an entire week. No further restrictions were imposed and patients had unlimited access to their personal possessions. This programme is even more lenient than the one we have reported previously (Touyz *et al*, 1984).

Treatment was undertaken by a multidisciplinary team comprising a consultant psychiatrist, a clinical psychologist, a dietitian, a family therapist, an occupational therapist, and skilled nursing staff. Minimum recommended weights were determined by the consultant and patients were required to gain weight to at least a body mass index of 18.5. The number of calories per meal was decided by the dietitian attached to the unit and was reviewed on a regular basis. Patients ate their meals in the dining room and all meals were supervised by the nursing staff. Besides dietary counselling, patients received supportive psychotherapy, and attended group therapy and occupational therapy.

The first 15 patients were weighed in their pyjamas daily at 7.00 a.m. whereas the next 15 patients were weighed at the same time each morning but only on Mondays, Wednesdays and Fridays. Patients were told their exact weight immediately after they were weighed.

Results

There were no significant demographic or clinical differences between the two groups. Patients who were weighed daily gained 0.19 kg per day whereas those who were weighed only three times per week gained 0.17 kg per day (Table I). This difference was not statistically significant ($t = 0.09$, $P = 0.93$). Furthermore, there was no significant correlation between age and rate of weight gain in those patients weighed daily ($r = 0.123$). Finally, the body mass index on admission did not correlate significantly with rate of weight gain in patients weighed daily ($r = -0.29$) or those weighed three times per week ($r = -0.35$).

Discussion

Vandereycken & Meermann (1984) have described their in-patient programme for refeeding patients with anorexia nervosa. Patients are initially weighed daily, but graduate to being weighed three times per week once they have gained a specified amount of weight. During the final phase of refeeding they are weighed only twice per week. However, if patients fail to meet certain requirements such as gaining at least 700 g per week, they are placed on the 'exception' programme, with their weight once again recorded daily.

Anderson (1985) weighs his patients three times per week but they have to stand with their back to the scale so as not to observe their actual weight. He argues that allowing patients to monitor their own weight increases their anxiety. There have been no published studies which support this conclusion. It has been argued that as much feedback as possible should be provided during treatment to facilitate weight gain and Anderson's approach would be contrary to this.

The results of this preliminary study revealed no significant differences between the rate of weight gain in those patients who were weighed daily and those who were weighed three times per week. This finding has important clinical implications in that it suggests that weighing patients less frequently does not adversely influence the rate of weight gain.

Once patients have reached their goal weight and have reduced their calorific intake accordingly, we introduce the concept of 'range' weighing. We prefer not to give our patients a target weight but rather a weight range of several kilograms, the lower end being the lowest acceptable weight (corresponds to a body mass index of 18.5). We set the scale at the lowest acceptable weight and then at a weight 2 kg higher. This enables us to document whether an individual patient is maintaining her weight within an acceptable range without focusing upon a specific weight. We only determine the patient's actual weight if her weight is found to be above or below the range agreed upon.

This technique provides reassuring feedback to the patient that she is able to control her weight within an acceptable range while on a maintenance diet. Neither the staff nor the patient are aware of an actual weight and the patient is encouraged not to worry about minor fluctuations in her weight. She is told that most persons show fluctuations of 1 kg or more in their weight and that this is quite normal.

In conclusion, the results of the present study suggest that the rate of weight gain in patients with anorexia nervosa during refeeding is not compromised by less frequent weighing. Furthermore, this may reduce the iatrogenic danger of intensifying the already heightened overconcern that these patients have with minor fluctuations in body weight.

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Anorexia Nervosa in an 80-Year-Old Woman

S. G. GOWERS and A. H. CRISP

A woman aged 80 years had suffered with anorexia nervosa in adolescence and the condition was rekindled after 50 years of remission. The remission was seen in the light of affective disorder and a chronic grief reaction.

Recently, two reports have been published on anorexia nervosa in middle and old age (Hsu & Zimmer, 1988; Rammell & Brown, 1988). Hsu & Zimmer suggest that eating disorders are becoming more prevalent in old age, because of a dramatic increase in the incidence of eating disorders as a whole, with a proportion surviving as lifelong chronic cases and because older women are beginning to succumb to social pressures to be thin. Kellett *et al* (1976) had previously reported the case of a 52-year-old widow who developed classic anorexia nervosa in relation to the marriages of her daughters. She was preoccupied with the death of her husband eight years previously, but was not thought to be suffering with a primary depressive disorder (never scoring above 11 on the Hamilton scale and denying depressive symptoms including loss of appetite), and she failed to respond to imipramine. Launer (1978) described a 70-year-old anorectic with obsessional neurosis. She was not thought to be depressed but no detail is given of bereavements. She failed to respond to clomipramine given for her obsessional symptoms.

Dally (1969) has attempted to explain the occurrence of anorexia in people aged over 35. He sees it as a variant which he terms 'anorexia tardive', and relates the condition to the subject's stage of life, and suggests that anorexia tardive might arise out of a resentful dependence on others or as a reason for retiring to bed after a lifelong history of pain or minor illness.

It is likely that as some anorectics do not shed their illness at the end of their reproductive life (Theander, 1985), some will remain with their condition into old age. However, one of Hsu & Zimmer's five cases and that of Rammell & Brown had no previous evidence of an eating disorder before a post-menopausal onset.

Crisp (1980) has suggested that late-onset anorexia nervosa, including that arising after the menopause, might represent, in the predisposed person, an attempt to achieve control, through avoidance of further decay, during a time of mounting personal uncertainty – that is, it is a response to the recognition of inner bereavement which has destabilised a long-standing fragile adolescent narcissistic adjustment. Such a response might also be activated or reinforced by external loss, for example, of a parent or spouse.

Case report

The subject, an 80-year-old widow, contacted one of the authors (AHC) after a radio programme about eating disorders. She believed herself to have anorexia nervosa and wanted an opinion, but did not wish to enter treatment.

Born in 1908, she developed anorexia nervosa at the age of 15 shortly after her menarche. In the patient's opinion social pressures to be thin, particularly from women's magazines (which she believes to have contained more dieting articles in 1923 than in 1988) combined with family factors to precipitate anorexia nervosa. Her father, a headmaster, strove to direct his children towards a university education, while sexual matters were strictly taboo.

She recalls her pride at losing weight from 57 kg to below 33 kg by abstinence and occasional laxative abuse and her fear when contemplating weight gain. She developed secondary amenorrhoea. Bingeing and vomiting were not features. She missed a complete year of school between the ages of 15 and 16, became regressed and childlike, and shared her mother's bed, refusing all food. Her medical attendant had diagnosed consumption. When the patient was 16, her obese mother went to stay for several months with her maternal grandmother, who was dying of cancer, and while the mother was away, the patient's disorder remitted and her weight rose to over 50 kg.

She left home to study at university at the age of 18 and while maintaining a normal weight, continued to avoid identifiable carbohydrate food and chocolates. She met her future husband while a student and believes that his non-judgemental acceptance of her was largely responsible for her full recovery. She took up work as a teacher and did not return home after university, gaining in confidence in her relationship with this man, who was five years her senior. She married at the age of 25 and from the point of view of her anorexia nervosa, the years until her menopause at the age of 48 were unremarkable, except that having risen to a weight of around 63 kg by the age of 28 she lost 3 kg with each of her four succeeding pregnancies. She had no further menstrual irregularities.

Her husband died when the patient was 65, and this appears to have led to a re-emergence of her anorectic attitudes. Some months after his death, having been depressed with complaints of early morning wakening and loss of appetite, though not weight loss at this stage, she took an overdose of barbiturates and alcohol and was admitted to a psychiatric hospital. She refused electro-convulsive therapy but after a short admission was discharged and has received no psychiatric treatment since. She considers that the depression lifted with supportive help from her general practitioner over the next year. The affective distress also lessened as her anorexia nervosa recrudesced. For the past ten years she does not consider herself to have been depressed. Since then, she has lived on a diet of fruit and vegetables with occasional lean meat or fish. She attempts to cut out all fat and sugar from her diet and takes regular laxatives in a conscious and readily acknowledged effort to control her weight, which she maintains at 79% of mean matched population weight (weight 45.8 kg, height 1.63 m). Her lifetime weight biograph is shown in Fig. 1.

Within the family there is a history of eating/weight disorder. Her mother was massively obese. A male nephew of the patient, who was brought up by the patient's parents, and one of the patient's daughters have suffered with anorexia nervosa. Another daughter and two grandchildren show some anorectic features and preoccupation with

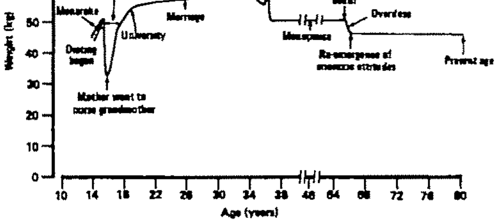

FIG. 1 Weight biograph.

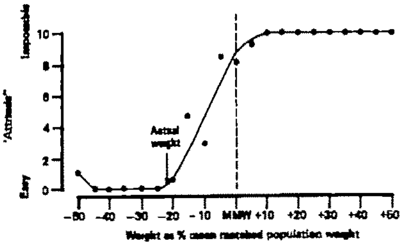

Fig. 2 Subject's attitudes to living at different hypothetical weights.

certain foods, though maintaining a low normal weight. Four other first-degree relatives suffer with asthma, eczema and psoriasis, which appear to have been particularly sensitive to stressful life events. There is no family history of depression or other psychiatric disorder.

At interview, the patient was open and forthcoming about her symptoms. Her attitude reflected the ego-syntonic nature of her symptoms, while her lack of defensiveness or denial is probably explained by her expectation that she would not be coerced into treatment. Her phobic avoidance of normal body weight is illustrated by her responses to a questionnaire on her attitudes to her ability to live at different hypothetical weights (Fig. 2). The shape of the response curve closely matches that of the pooled results of an anorectic population (Crisp *et al*, 1986), in that her aversion to mean matched weight is almost as marked as her attitude to obesity.

Her manner suggested an extreme attention to detail and she thought that her preoccupation with neatness and order at home and a tendency to parsimony related to her anorectic state. She was financially very comfortable, was sprightly and drove her own car, suggesting no immediate threats to her security or independence. There was no evidence of depressed mood, tearfulness or suicidal ideology. Sleep was unimpaired and she retained her appetite and preoccupation with food. She was able to discuss her husband without distress and was no longer preoccupied with loss. She maintained a quiet social life but kept in close and active contact with her extended family. Cognitively, she was remarkably well preserved for her age, with good long-term and short-term memory and calculating abilities, and she was exceptionally well informed of current events.

Discussion

Anorexia nervosa is a disorder of which the cardinal symptoms are behavioural (self-induced weight loss which is not the result of loss of appetite), psychological (phobic avoidance of normal body weight), and endocrine (chiefly a reduction in the output of gonadotrophins with amenorrhoea in women) (Crisp, 1980). Associated features which may be present are an overconcern with food and nutrition, bingeing, conscientiousness, obsessional and depressive symptoms, and physical symptoms resulting from emaciation. Characteristically a disorder of adolescence, anorexia nervosa has been viewed as an avoidance stance, providing a maladaptive solution to an existential problem (often related to puberty), sooner or later afflicting the subject and her family (Crisp, 1980).

Retrospective assessment would suggest that the subject fulfilled the above criteria for anorexia nervosa at the age of 15 and her self-reports of the social and family precipitants were typical. There is considerable evidence for a current diagnosis of anorexia nervosa in the subject now aged 80, who we believe to be the oldest case reported. She shows many of the psychological and behavioural features, while endocrine changes are not applicable. Why should this patient develop anorexia nervosa at this stage in her life?

It would appear that after her husband's death, she suffered a depressive reaction with suicidal intent, from which she fully recovered in terms of her subjective mood and biological symptoms, including her appetite. Depression and grief are common themes in reports of anorexia nervosa in the elderly. Indeed, the extensive review by Swift *et al* (1986) of the relationship between affective disorder and eating disorders failed to resolve the link between these conditions at any age. Three of Hsu & Zimmer's (1988) five elderly patients were either depressed at presentation or had a history of major depression. Rammell & Brown's (1988) case had suffered three major losses including that of her husband. She felt that she had not been able to grieve fully and had suffered episodic depression since. She did not suffer loss of appetite, had an intense fear of gaining weight and failed to respond to antidepressants, although she recovered with weight restoration and management of her eating disorder.

Parkes (1980), in an extensive review of his own and other studies of grief, describes the essential features as a preoccupation with the dead person, with tearfulness and extreme distress with any reminder of them. Our patient was not distressed discussing her husband. Further, Parkes states here that a case of nine and a half years is the longest grief reaction he has seen. Our patient was bereaved 15 years previously. Where weight loss is present in chronic grief, this is seen as being due to loss of appetite or a loss of interest in food related to a preoccupation with the 'searching' feature of grief in which the essential needs of the subject are neglected. In an earlier study, Parkes (1964) reviewed 94 cases of admission to the Bethlem Hospital for psychiatric treatment within six months of a bereavement: anorexia nervosa was not the presenting condition in any.

How can one link an adolescent onset of anorexia nervosa with concerns about pubertal development, the patient's perception of dynamics operating within her family of origin, and her relapse into anorexia nervosa in old age, with her history of grief and depression as frequently reported in late-onset cases? Is her recovery from both her depression and her grief reaction incidental, or related to her present psychopathology? A connection may be provided by the concept of the 'life review'.

Butler (1963) saw the reminiscences of the elderly as representing part of a normal process precipitated by the realisation of mortality and approaching death. The life review is characterised by a progressive self-reflection of past experiences and a resurgence of unresolved conflicts. If the life review is completed satisfactorily, new solutions to earlier difficulties will be integrated, giving new meaning to one's life. If, however, such conflicts are too painful, or insoluble, it may be necessary to defend against their intrusion into consciousness.

Crisp has always seen anorexia nervosa as providing an adaptive biological solution to an existential conflict and we would see the re-emergence of our subject's anorexia nervosa as an attempt to utilise a previously discovered coping strategy to keep negative emotions at bay (in this case panic and depression rooted in her long-standing sense of insecurity, reactivated by her husband's death). Her control of her body weight could be seen as giving her a sense of purpose and single mindedness, thereby reducing to the point of exclusion once again her emotionality at a time of personal uncertainty.

We would echo Hsu & Zimmer's (1988) plea that clinicians who care for the elderly consider the possibility of an eating disorder in the differential diagnosis of the patient with weight loss or vomiting. Anxieties about weight should be further investigated by careful history taking and, as in the younger subject, useful valuable information may be provided by a relative.

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Psychosis Following Use of Proprietary Antidiarrhoeal Medicines

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Anticholinergic psychosis was observed to follow ingestion of proprietary antidiarrhoeal preparations by a 63-year-old woman. Possible abuse or accidental overuse of such medicines in the acutely psychotic patient should always be considered.

Anticholinergic psychosis due to both intentional and unintentional abuse of anti-Parkinsonian agents is well recognised. A similar toxic psychosis due to abuse of over-the-counter sedatives has also been documented (Gardner & Hall, 1982). The relationship between psychosis and abuse of proprietary medications has been previously described, particularly with reference to sympathomimetic agents as constituents of nasal decongestants (Lambert, 1987), and oral anorectic agents (Schaffer & Pauli, 1980). This report describes a case of anticholinergic psychosis following unintentional overuse of proprietary antidiarrhoeal preparations.

Case report

A 63-year-old single lady with no previous psychiatric history was admitted to a psychiatric hospital, having been found wandering by the police earlier that day. On admission she was paranoid, agitated and actively hallucinating. She experienced second-person auditory hallucinations, and explained to the doctor "Mrs Cox is talking to me now, she is saying such dreadful things, you know – listen!" She also experienced visual hallucinations, and stated "I saw a sheep's head in the bed here beside me before you arrived". At a later time she said that on her first day of admission she had been terrified by seeing a row of men in descending order of height dancing in front of her. She also experienced gustatory hallucinations and stated "I just drank some water doctor. Don't drink it. It was full of excrement and I could taste it doctor." Her thought was clearly disordered and her speech at times incoherent. She was fully orientated in time, place and person. Her concentration was poor and she was distractible. The assessment of her memory and the remaining higher mental functions was not possible owing to the intensity of her hallucinations. Physical examination revealed a tachycardia and slow pupillary light reflex responses. Neurological examination was otherwise normal.

An organic cause for her psychosis was suspected and a comprehensive haematological investigation, drug screen, electroencephalogram, and cranial computerised tomography scan were arranged. She received chlorpromazine (50 mg) on the morning of her admission. No further treatment was prescribed and her psychosis resolved spontaneously over the next 48 hours. During that period she remained restless and actively hallucinating. Disorientation in time and place were noted on only a single occasion. She was perplexed by what had happened and stated "I never had a very vivid imagination but over those last few days, I had a cast of hundreds". She described how, in the week before admission, she had suffered bouts of diarrhoea. She obtained antidiarrhoeal preparations from the pharmacist. Over the two days before admission she ingested 30 tablets of Diocalm, a preparation containing morphine hydrochloride (0.395 mg per tablet). She also took an unspecified quantity of a preparation called Enterosan, which contains morphine hydrochloride (0.275 mg) and belladonna extract (1.8 mg) per tablet. Repeated physical, laboratory and radiological investigations were all normal and the final diagnosis was that of an anticholinergic psychosis due to the belladonna in the antidiarrhoeal preparation. At two-year follow-up she remains very well.

Discussion

Organic psychoses have been associated with drugs with anticholinergic properties, such as neuroleptics (Hvizdos *et al*, 1983), tricyclic antidepressants (Goodwin, 1983) and anti-Parkinsonian agents (Woody & O'Brien, 1974). Psychotic states in clear consciousness may follow use of hallucinogens, central stimulants, dopamine agonists, β-adrenergic-blocking drugs, and opiate analgesics (King, 1986). The peripheral features of anticholinergic toxicity include cycloplegia, mydriasis, reduced gastrointestinal secretion and smooth-muscle motility. Among the central features, depression, elation, hallucinations and delirium are common. There may be difficulty differentiating from other organic psychoses or acute schizophrenic psychoses. Cutting (1987) reported that visual hallucinations occurred in 34% of his patients with an organic psychosis and in 6% of those patients with a schizophrenic psychosis. Physostigmine, a non-quaternary anticholinesterase, has been reported as an effective antidote in treatment of anticholinergic psychosis (Allen *et al*, 1979), at an initial dose of 1–3 mg parenterally. However, most cases resolve spontaneously over two days.

Compounds containing atropine-like substances are widely available to persons without prescriptions in eye-drops, antiemetics and antidiarrhoeal preparations.

Many patients take such medications without any awareness of potential side-effects. While this patient had taken a large quantity of Diocalm, the clinical presentation, accompanied by Lilliputian hallucinations, supports a diagnosis of acute anticholinergic toxicity from the Enterosan preparation. Some interaction between both compounds cannot be ruled out although psychotic states due to interaction between opiate-containing drugs and drugs with anticholinergic properties have not been documented (Committee on Safety of Medicines, personal communication). Similarly, there have been no previous reports of psychosis in association with antidiarrhoeal preparations (Committee on Safety of Medicines, personal communication). In addition to earlier accounts of psychosis following misuse of other proprietary medicines (Schaffer & Pauli, 1980; Gardner & Hall, 1982; Lambert, 1987) this case stresses the importance of eliciting details pertaining to use of over-the-counter medications (in addition to prescribed and illicit drugs) when evaluating the acutely psychotic patient.

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Neuroleptic-Induced Priapism, Hepatotoxicity and Subsequent Impotence in a Patient with Depressive Psychosis

ERIC Y. H. CHEN and ALAN S. LEE

A 59-year-old man experienced priapism five days after taking neuroleptics and abnormal liver function after 28 days. These side-effects are possibly explained as hypersensitivity reactions to the drug.

Priapism is a rare but serious side-effect of neuroleptic medication. It involves persistent turgidity of the penile corpora cavernosa in the absence of sexual arousal. The corpus spongiosum and the glans are spared. Reports of drug-induced priapism in psychiatric patients are becoming increasingly frequent, but the underlying mechanisms remain largely unknown (Kogeorgos & de Alwis, 1986; Eikmeier, 1987). Much of what is known has been derived from laboratory animal studies (e.g. Abber *et al*, 1987), but their clinical relevance is still unconfirmed. Meanwhile, case studies continue to provide insights into the nature of drug-induced priapism, particularly when it is associated with other rare side-effects.

Case report

A 59-year-old driver with no significant history of previous medical or psychiatric illness was admitted with an agitated psychotic depression. Investigations including full blood count, erythrocyte sedimentation rate, urea and electrolytes, liver-function tests, thyroid-function tests, chest radiography, serology and autoantibody screen were all normal. On the first day of admission, he was given chlorpromazine (200 mg) intramuscularly because of agitation. He received no further medication but five days later he developed a persistent erection. A diagnosis of priapism was made and he underwent an emergency decompression and a shunt operation.

Subsequent investigations included computerised tomography, and demonstrated a small area of low attenuation in the right caudate nucleus and mild cortical sulcal widening. Studies of cerebrospinal fluid were normal apart from a mildly elevated immunoglobulin level.

The patient remained mentally unwell. On the 28th day his liver functions, which were normal on admission, were noted to have become deranged. Gamma glutamyltranspeptidase was elevated to 150 units (normal range 11–51), alkaline phosphatase 723 (98–280), alanine aminotransferase 525 (0–40), and bilirubin 16 (0–17). These gradually improved and returned to normal within the next three weeks.

Subsequently, the patient received a course of electroconvulsive therapy (ECT), which was ineffective. He was then treated with lithium and his mental state gradually improved. His recovery from the psychosis was complicated by an awareness of loss of potency. His feelings of loss and uncertainty over his sexual role intermingled with underlying depressive feelings of guilt and self-doubt. These were particularly difficult to manage as he had no recollection of the events leading to the operation. Five months after admission, he was discharged to a day hospital from where he subsequently returned to work.

Discussion

This case offers two interesting observations, one on pathophysiology, the other addressing the psychological sequelae of neuroleptic-induced priapism.

Priapism and impairment of liver function are both fairly uncommon side-effects of neuroleptic drugs. Reports of priapism include those of Fishbain (1984), Eikmeier (1987) and Kogeorgos & de Alwis (1986). Impairment of liver function is estimated by Sherlock (1985) as occurring in 2% of patients who take neuroleptic drugs.

It is widely assumed that priapism involves relaxation of cavernous and arteriolar smooth muscles, leading to increased blood flow into the sinusoidal spaces, and compression of the emissary veins against the tunica albuginea with a consequent decrease in venous outflow (Lue *et al*, 1986). Brindley (1983) showed that alpha-adrenoceptor blockers injected into the human corpus cavernosum cause erection, and Abber *et al* (1987) argued from experiments on dogs that the priapism occasionally caused by chlorpromazine is due to its blocking of alpha-adrenoceptors. Case reports which show drug-induced priapism resolving with local or intravenous application of anticholinergic medication have led to the suggestion that parasympathetic dominance is also contributory (Osborne, 1974; Fishbain, 1984). These observations and hypotheses are linked by the possibility that parasympathetic dominance results from alpha-blockade within the sympathetic system.

Such models are relevant insofar as they approximate to clinical situations. Timing is of particular importance. Whereas in the experimental paradigm, priapism occurs within minutes of introduction of the inducing agent, clinical case reports cite periods of one day to nine months between the initiation of medication and the associated priapism (Fishbain, 1984; Kaisary & Smith, 1986*a*,*b*; Kogeorgos & de Alwis, 1986). This variability of latency argues against the alpha-blockade hypothesis. In our patient, priapism occurred five days after a single intramuscular injection of chlorpromazine, which again suggests that the alpha-blockade model is inadequate.

In addition to psychotropic medication, priapism is also associated with alterations in blood viscosity, blood dyscrasias and local pathology of the pelvis. Clearly the mechanisms involved in these situations are likely to be diverse. Apart from alpha-blockade, thrombosis, hypotension, mechanical blockage and extrapyramidal effects have been suggested as pathogenic factors (Eikmeier, 1987).

In neuroleptic-induced priapism, Korgeorgos & de Alwis (1986) suggested that a causative role for anticholinergic and hypotensive properties of neuroleptic drugs was unsubstantiated, and proposed that priapism was a complex and multifactorial disorder requiring the interaction of several factors.

A further promising way of exploring the underlying mechanisms is to study the association between the occurrence of priapism and the pattern of other side-effects of the offending agent. For instance, if it is postulated that priapism occurs because of vulnerability to the antiadrenergic effect of a neuroleptic, other antiadrenergic side-effects such as postural hypotension would be more likely in the same individual. Unfortunately, case reports seldom supply information on coexisting side-effects apart from one account, where a concurrent acute dystonic reaction was reported (Fishbain, 1984). In our patient, there was no evidence of side-effects due to alpha-blockade; rather, priapism was followed by another rare side-effect of neuroleptic medication, hepatotoxicity.

It is generally accepted that chlorpromazine-induced hepatotoxicity is most likely to be detected two to four weeks after the introduction of medication, and involves a hypersensitivity reaction (Sherlock, 1985). This leads us to wonder whether in our patient, susceptibility to hypersensitivity reactions with chlorpromazine may have contributed to both priapism and hepatotoxicity.

It is acknowledged that in most drug reactions, the role of hypersensitivity is difficult to prove. However, hypersensitivity is strongly suggested if the following criteria are fulfilled (Terr, 1982):

(a) a reaction occurs in a small proportion of exposed individuals
(b) there is a latent period between exposure and reaction
(c) a reaction is elicited by relatively small doses
(d) there is association with other known hypersensitivity phenomena (e.g. eosinophilia).

Previous reports (Fishbain, 1984; Kaisary & Smith, 1986*a*,*b*; Kogeorgos & de Alwis, 1986) have shown that neuroleptic-induced priapism satisfies criteria (a), (b) and (c). The fourth criterion is addressed in the present case which reports an association between chlorpromazine-induced priapism and hepatotoxicity. This leads to a proposal that hypersensitivity may be one of the mechanisms involved in some cases of neuroleptic-induced priapism.

One difficulty with our hypothesis is a difference in timing of the priapism (day 5) and the detection of impaired liver function (day 28). It is noted that upon detection on day 28, the liver function improved continously until it returned to normal in the next three weeks. This suggests that impairment of liver function probably had already peaked before day 28. Furthermore, in contrast to a generalised hypersensitivity reaction, local hypersensitivity involves the interaction between antigen and host immune system (both humoral and cellular) at local tissue sites (Terr, 1982). As this depends on kinetic factors specific to the organs involved, exact concurrence in timing would not be expected in reactions at two separate sites.

One further interesting aspect of this case concerns the pattern of hepatotoxicity. Whereas the classic picture of chlorpromazine-induced hepatotoxicity involves cholestatic jaundice, subclinical impairment of liver function has been described and is probably not uncommon (Sherlock, 1985). Because of the absence of clinical jaundice, impairment of liver function in cases similar to this one could have remained undetected.

Whatever the underlying mechanisms, there are important physical and psychological consequences of neuroleptic-induced priapism. Priapism is an indication for urgent urological intervention. Even with treatment, only 8 out of 22 patients in one series retained potency (Kaisary & Smith, 1986*b*). A difficult problem can arise when a psychotic patient develops neuroleptic-induced priapism, receives treatment in the acute phase of his illness, and then gradually recovers from the psychosis to discover a loss of potency. In the existing literature, few authors have addressed this aspect of management. In one case report, Griffith & Zil (1984) described mild depressive symptoms in their patient and drew attention to the emotional reactions following loss of potency. In our patient, the awareness of loss of masculinity was incorporated into a complex system of cognition concerning guilt and worthlessness. This occurred as he emerged from a deeply psychotic state and began to re-establish contact with reality. Compounded by amnesia concerning the priapism and subsequent treatment, adjustment to the disability was very difficult and required repeated counselling. Staff awareness and experience of the situation was limited by the small number of cases previously encountered in the general psychiatric practice. Further work in this area is required so that knowledge could be shared and expertise pooled to help those involved to cope with an additional humiliating and traumatic disability on top of a psychiatric disturbance.

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Fluvoxamine/Pimozide Treatment of Concurrent Tourette's and Obsessive–Compulsive Disorder

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GEORGE R. HENINGER and DENNIS S. CHARNEY

A 25-year-old man with a history of Tourette's syndrome presented for treatment of OCD symptoms. Fluvoxamine worsened tics, led to coprolalia, and did not help the OCD. The addition of pimozide dramatically reduced both OCD and Tourette's symptoms. Double-blind sequential discontinuation of fluvoxamine and pimozide confirmed that pimozide alone reduced only tics and the combination of fluvoxamine and pimozide was required for the improvement in OCD. Tics may reflect a subtype of OCD. Some OCD patients unresponsive to a 5-HT reuptake inhibitor alone may benefit from the addition of a dopamine antagonist.

The efficacy of drug treatment for obsessive–compulsive disorder (OCD) has now been established (Insel & Murphy, 1981). In double-blind trials, the potent serotonin (5-HT) reuptake inhibitors chlorimipramine (Insel & Murphy, 1981) and fluvoxamine (Goodman *et al*, 1989*a*) were significantly better than placebo in reducing the symptoms of OCD patients, whereas tricyclics with less effect on 5-HT reuptake inhibition such as nortriptyline (Thoren *et al*, 1980) and desipramine (Leonard *et al*, 1988) were largely ineffective. These findings lend support to the hypothesis that the 5-HT system plays a role in the pathophysiology of OCD. However, as many as 40–50% of OCD patients do not derive clinical benefit from 5-HT reuptake inhibitors (Insel & Murphy, 1981; Goodman *et al*, 1989*a*). This suggests that OCD may be heterogeneous, and has stimulated interest in identifying possible clinically and biologically distinct subtypes.

One possible subtype of OCD is represented by those patients with dual diagnoses of OCD and chronic multiple tics (CMT) or Tourette's syndrome (TS). Approximately 50% of TS patients have OCD symptoms (Pauls *et al*, 1986), and recent studies of first-degree relatives of TS probands suggest a possible genetic relationship between TS and OCD (Pauls & Leckman, 1986). TS is thought to involve dysfunction of brain dopamine systems, because of the effectiveness of neuroleptic drugs, such as haloperidol and pimozide, in reducing the severity of tics in most patients with TS (Shapiro, 1976). Monoamine reuptake inhibitors, such as imipramine (Sverd, 1988) and chlorimipramine (Caine *et al*, 1979), have been reported to exacerbate tics.

To our knowledge, there is only one published report of the response of OCD symptoms in patients with TS to treatment with potent 5-HT reuptake inhibitors (Riddle *et al*, 1988). Therefore, it was of interest to us to evaluate treatment response to fluvoxamine in a patient with a lifetime history of TS and recent-onset OCD.

Case report

A 25-year-old single white man with a history of simple phonic (e.g. coughing, barking, and grunting), simple motor (e.g. blinking), and complex motor (e.g. hitting nose) tics since seven years of age was referred to our OCD clinic for treatment of severe obsessions and compulsions. About 18 months before referral he had experienced the gradual onset of increasingly severe obsessions concerning symmetry, exactness and order, and fear of acting on unwanted aggressive impulses. He also complained of a compulsive need to write things down, to check repeatedly, and to order and arrange various items. He was no longer able to work and was continuously suffering from obsessional thoughts and performing rituals.

He had received no neuroleptics for treatment of his tics from age 11 to 23 and the severity of his tics had remained mild over the years, not interfering with his functioning. However, as the OCD symptoms worsened, he noted a flare-up of the tics so that they were now very noticeable and were interfering with his social functioning. In the year before referral, he had also begun to experience increasing unhappiness, difficulty falling asleep (because of obsessive thoughts and compulsions), loss of appetite, difficulty with concentration, loss of self-esteem, and hopelessness. He attributed these depressive symptoms to the severe OCD and to the difficulty he was having in functioning.

Before referral he was treated with haloperidol (2 mg/day) but had a severe dystonic reaction on the third day of treatment. Thioridazine (50 mg/day) and trihexphenidyl (2 mg/day) were substituted, resulting in a reduction in agitation but minimal effects on the obsessions or compulsion. These drugs were discontinued after two months and alprazolam (1.5 mg/day) was initiated, with a similar decrease in anxiety and agitation but no reported effects on the OCD. The patient continued to receive this dose of alprazolam while in a supportive group-therapy programme for one year without improvement until referral to our clinic.

On presentation to the OCD clinic he demonstrated no evidence of a formal thought disorder, perceptual abnormalities, or cognitive impairment. Physical and routine laboratory examinations revealed no abnormalities. After giving written informed consent, he participated in a randomised, parallel-group, double-blind study comparing fluvoxamine and desipramine (Goodman *et al*, 1990). Figure 1 presents his scores on the Yale–Brown Obsessive Compulsive Scale (Y-BOCS; Goodman *et al*, 1989*b*) and on the Yale Global Tic Severity Scale (GTS; Leckman *et al*, 1988) throughout his treatment. After eight weeks of double-blind medication, OCD symptoms worsened modestly (Y-BOCS: 34, baseline; 38, week 8, Fig. 1) and tics had worsened (GTS: 20, baseline; 25, week 8, Fig. 1) so that he began to experience coprolalia for the first time in his life. At that time the study concluded and the code was broken. On breaking the code, he was noted to have received fluvoxamine with the dose being increased to 150 mg/day by the third week and 250 mg/day by the fifth week and maintained at a constant level thereafter. Given the lack of clinical response and the worsening of tics, pimozide (1 mg/day) was added in an open fashion to ongoing fluvoxamine. Over the next four weeks both OCD symptoms and tics diminished dramatically, so that he was spending less than one hour a day ruminating or performing compulsive rituals, and the tics were barely noticeable.

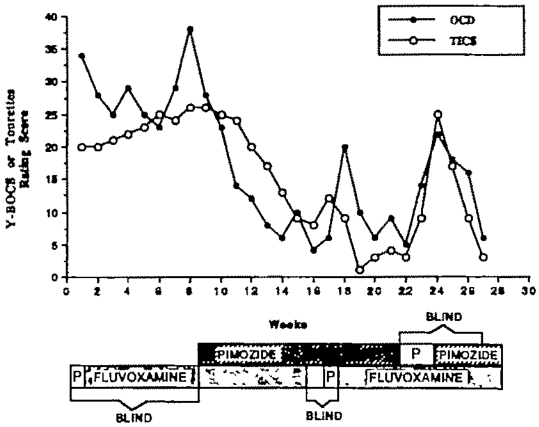

FIG. 1 Yale–Brown Obsessive Compulsive Scale (Y-BOCS) and Yale Global Tic Severity Scale during course of treatment (●—● OCD, ○—○ tics). (P = placebo, blind refers to double-blind portion of treatment, pimozide dose 1 mg/day, fluvoxamine dose 250 mg/day from week 5.)

In order to clarify the respective roles of fluvoxamine and pimozide in the observed clinical improvement, a double-blind discontinuation of fluvoxamine was begun on week 7 of combined fluvoxamine/pimozide treatment and a double-blind discontinuation of pimozide initiated on week 15 of combined fluvoxamine/pimozide treatment (Fig. 1). The fluvoxamine-discontinuation phase began with active fluvoxamine (250 mg/day) during the first week followed by tapering doses of fluvoxamine: 150 mg/day for one day; then 50 mg/day for one day; then placebo fluvoxamine. During the pimozide-discontinuation phase the pimozide was simply discontinued and substituted by placebo on the first week. All medication during the double-blind discontinuation phases for both fluvoxamine and pimozide were administered with medication kits containing medication doses in individual daily packets for the entire week. The patient and treating physician were blind as to the medication contained in the kits.

During the discontinuation of fluvoxamine, phonic and motor tics did not change, but the patient experienced an acute exacerbation of OCD symptoms requiring that the code be broken and active fluvoxamine reinstituted. The OCD rapidly improved to previous low levels within one week (Fig. 1). During the discontinuation of pimozide a more gradual worsening of both tics and OCD ensued in the two weeks following pimozide discontinuation (Fig. 1).

Active pimozide (1 mg/day) was restarted blind and a gradual improvement to previous levels occurred over the next three weeks. In the eight months since this study, the patient has remained greatly improved on the same regime, with a significantly increased appetite, carbohydrate craving, and a 7 kg weight gain (4 kg heavier than any previous weight) being the most notable side-effects.

Discussion

The combination of the selective and potent 5-HT reuptake inhibitor fluvoxamine with the potent dopamine antagonist pimozide, led to a near remission of OCD symptoms and a reduction of tics. Fluvoxamine alone appeared to exacerbate tics, leading to the new onset of coprolalia, without improving OCD symptoms. Pimozide alone reduced tics very slightly. The lack of response of OCD symptoms to pimozide alone in this case is consistent with the widely held clinical impression that neuroleptics alone are rarely beneficial in OCD (Insel & Murphy, 1981).

In this patient, the combination of fluvoxamine and pimozide appeared to be necessary for clinical improvement of OCD symptoms, suggesting that both the dopamine and 5-HT systems were involved in the observed clinical response. Studies of laboratory animals suggest that there is a complex interaction between 5-HT and dopamine systems in the brain (Gabay, 1981). Central 5-HT systems are believed to maintain a tonic inhibitory tone on the dopamine system (Korsgaard *et al*, 1985). Paroxetine, a potent 5-HT reuptake inhibitor, decreases the amphetamine-induced increase in stereotyped behaviour and increases haloperidol-induced dystonia (Korsgaard *et al*, 1985). In contrast, cyproheptadine, a 5-HT antagonist, increases amphetamine-induced stereotyped behaviour and decreases haloperidol-induced dystonia in monkeys (Korsgaard *et al*, 1985).

The degree of weight gain on recovery in this patient was unexpected. The description by the patient of an increase in appetite and craving for carbohydrates is at odds with the absence of weight gain usually seen with fluvoxamine. The relationship of the weight gain to an antidepressant response in this case remains unclear. Possible explanations for the weight gain could include a decrease in motor activity due to a reduction in ritualistic behaviour, improved mood, or an atypical neurochemical effect of the combination of these two drugs on appetite-regulating mechanisms of the central nervous system. The fact that the patient's weight increased far beyond any previous weight and the description by him of carbohydrate craving argue for some central change in appetite resulting from the combination of fluvoxamine and pimozide.

The present case suggests that some OCD patients unresponsive or partially responsive to a 5-HT reuptake inhibitor alone may respond after the addition of a dopamine antagonist. Such patients may represent a distinct subtype of OCD in which symptoms are mediated by an abnormality in both the dopamine and 5-HT systems. However, the clinical implications of adding neuroleptics to 5-HT reuptake inhibitors in OCD need to be clarified by controlled trials in well defined populations of OCD patients (e.g. OCD with or without tics or family history of tics). This is especially important considering the potential risks of tardive dyskinesia with chronic neuroleptic treatment.

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Camptocormia

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Camptocormia is a rare conversion syndrome found mainly in soldiers from World War I and II. No case has been reported since, but this paper reports the syndrome in a former soldier of the Nicaraguan army. It is characterised by severe frontal flexion of the spinal cord and knees, with passive dropping of both arms, and sometimes behavioural abnormalities.

Conversion reactions have traditionally been one of the main causes of psychiatric morbidity in wartime, especially before 1950. Among allied troops in World War II they accounted for 10–25% of all psychiatric casualties (Davies & Bick, 1946) equally affecting male and female military personnel (Ballard, 1945) and being reported in different cultures. Horder (1939) classified 'war neuroses' in three groups: (a) 'general concussion reactions' in men without visible wounds (5–10%), (b) 'emotional shock' (acute) reactions (80%), and (c) "nervous and mental exhaustion as a result of prolonged strain and hardship" (10–15%), this latter group being close to the current concept of post-traumatic stress disorder. Although the available epidemiological data cannot be compared, it seems by analysing the amount of published literature that the profile of wartime casualties changed from being conversion disorders in World War I to acute anxiety reactions in World War II. This phenomenon could be partially substantiated by the shift from a preponderance of French psychiatry – and its interest in conversion and hysteria – in World War I, to more Anglo-American psychiatry, interested in the study of anxiety disorders and psychosomatic medicine.

Orthopaedic symptoms have been classic target symptoms in conversion. Boland & Corr (1943) found psychogenic rheumatism to be the major problem in 450 admissions among soldiers with a previous diagnosis of arthritis. Fox (1945) presented the results of a co-operative evaluation between a psychiatrist and a military orthopaedist in the assessment of postural malformations. They found psychogenic components in "the vast majority of cases". Sir Arthur Hurst has been the most prominent figure in this area. He described one of the first cases of camptocormia in England in 1918 (Walker, 1928) and kept on collecting evidence of all sorts of 'hysterical' contractures for more than 20 years (Hurst, 1941).

Camptocormia (from *kamptos*, curved, and *kormos*, trunk) constitutes a rare psychogenic syndrome characterised by a frontal flexion of the vertebral column with passive dropping of both arms and a variable degree of genuflexion, producing a simian appearance. Unsteady gait is also often found. The first reports date from when Souques & Rosanoff-Saloff made exhaustive descriptions of French soldiers suffering from the disorder (Rosanoff-Saloff, 1916; Souques, 1916). In 1928 Walker reports "about 50 case records" in the literature. The post-traumatic nature of the disorder and the existence of secondary gains is emphasised in the first records. Belgrano & Giordano (1947), in a description of three cases, consider true pain as the starting point of the disorder, with secondary anti-algic bending, although they drew attention to the possibility of camptocormia in the absence of any irritating stimulus and due to imitation or suggestion. No case of conversion camptocormia has been reported to our knowledge since the end of World War II (Outes, 1956). A case is presented here of a soldier in the Nicaraguan army suffering from the disorder.

Case report

JMM, a 36-year-old man, was brought, tied up, to the emergency room of the Manolo Morales Hospital (Managua) by members of his family. JMM took an active part in the 1979 rebellions before the final general uprising of the Sandinista Revolution. He fought in the army for four years, during which he had suffered four episodes of severe craneo-encephalic trauma. There is no reliable information about the first three episodes, but the last, which provoked as the sequelae the postural abnormality, was due to the explosion of a mortar close to him. The shockwave hurled him over several metres. He remained unconscious for about two hours. He was not hit by shrapnel either on this occasion or previously, but suffered global concussion.

JMM presented with a severe kyphoscoliosis. His trunk was inclined 70° forward and his knees were bent at approximately 15°. He walked with small steps and wide swings of his arms. The psychiatrist was called after a violent, apparently dissociative, outburst in the emergency room. He had been extremely aggressive and threatened his family with a large knife. His mother explained that since his last injury, JMM had had sudden outbursts of rage. He remained in bed most of the time. His answers were always verbally aggressive. His current posture had first appeared more than a year before. In spite of this, his family explained that he slept in a normal anatomical position.

The physical and X-ray examinations revealed no damage to the vertebral column. There were no painful points in his back or legs. No pain was elicited by passive movements. The patient would not answer any questions and simply shook his head slightly. He was diagnosed as suffering from a conversion disorder. Treatment was begun with carbamazepine (400 mg/day). An appointment was made for out-patient follow-up but neither the patient nor any family member returned.

Discussion

Camptocormia is a rare disorder with few descriptions in the literature. Hamlin (1943), studying a sample of 474 soldiers attending a general hospital between June 1941 and April 1942, reported 17 cases of conversion disorders, two of them being camptocormias. The two most important series available are the 19 cases published by Sandler (1947) between 1945 and 1947 and the eight cases collected by Sutro & Hulbert (1946). In both samples 70–75% of cases are reported in Negro soldiers. Patients are subjectively described in Sandler's sample as having great resentment and hostility to the point of paranoia, although they are superficially polite and friendly. Explosive outbursts of aggression are often found, a fact also described in some of Hurst's cases, but not by any other of the authors mentioned. Although 17 out of the 19 soldiers in Sandler's sample were married, impotence was often found, and two-thirds of the patients had no children. There was a family history of backaches or camptocormia in 10 cases (10/10 fathers, 3/10 mothers).

Treatment during World War I did not differ very much from the original one proposed by Souques (1916); a plaster corset, psychotherapy and anaesthetic infiltration. As Walker (1928) describes it: "The patient is stretched out on a flat board like a butterfly in a show-card and kept in that position for about 18 hours of the 24. He is securely fastened, and board and patient are bodily moved about from time to time in various planes . . . Gradually more and more time is spent in the vertical position." Reviews after World II (Sutro & Hulbert, 1946; Sandler, 1947) report improvement simply with some supportive psychotherapy and discharge from military service without any additional therapeutic measure.

As Anderson (1941) pointed out, the literature regarding conversion disorders merged under the same heading three different phenomena: "(1) conversion symptoms or reactions (2) transient abnormal psychic states with alterations of consciousness and (3) hysterical character". Current classifications distinguish these three aspects but focus conversion disorders in a different way. On the one hand, there is a general agreement that a hysterical personality is frequent (15–25% of cases) but not a necessary component of conversion disorders (Shalev & Munitz, 1986), with obsessive or schizoid personalities being found often, and normal personalities seeming to be the most frequent. On the other hand, some authors (Shalev & Munitz, 1986) point out that in a long-term follow-up, somewhere between 25% and 50% of all severe conversion disorders are ultimately diagnosed as suffering from some kind of neurological disorder, more than half being epilepsy. The classic definition of conversion disorders as a symbolic representation of physical conflict in terms of motor and sensory manifestations have to be reconsidered. The constellation of motor and behavioural symptoms apparently associated in camptocormia are striking. In the broad field of conversion disorders it constitutes an especially puzzling entity that is probably destined to disappear before it is fully understood.

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Paraphilic Infantilism

A Rare Case of Fetishistic Behaviour

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A rare case of infantile behaviour of a fetishistic nature in an 80-year-old man was possibly related to a head injury at the age of six, when his regression first began. Treatment was deemed unwarranted.

Fetishism was defined by Stoller (1976) as the need for inanimate or part objects for sexual arousal. The word 'fetishism' means the worship of inanimate objects, but when given a sexual connotation it may be defined (DSM–III) as "the use of non-living objects as a repeatedly preferred or exclusive method of achieving sexual excitement" (American Psychiatric Association, 1980).

In legal terminology 'paraphilia' is perversion or deviancy (Money, 1986). Deviant sexual behaviour, the dependence on unacceptable or unusual stimulus for sexual arousal and satisfaction, was reclassified as 'paraphilia' in DSM–III–R (American Psychiatric Association, 1987). Paraphilic infantilism is not given its own number in DSM–III–R and, therefore, should be classified as "Sexual disorder, 302.90 paraphilia not otherwise specified." The issue of its classification was discussed by Money (1984) when he recorded around 30 paraphilias. Paraphilic infantilism is also known as 'nepiophilia', which is like paedophilia except that the age range is restricted to infancy (from Greek, *nepon*, infant). The paraphilic condition of impersonating a baby is autonepiophilia, or paraphilic infantilism (Money, 1986), a case of which is reported below. To the author's knowledge, no similar case report has been documented.

Case report

This 80-year-old man was first referred for psychiatric opinion in 1977, following his arrest by the local police. He had been taken into custody for indecently exposing himself to a neighbour; at the time he appeared disturbed and anxious. The neighbour had been invited to his house on the previous day to examine some household effects. The patient then adopted a baby posture, inviting her to change his nappy and to feed him, after which he masturbated. He also visited her next day and exposed himself on her doorstep.

According to his general practitioner, his sister was admitted to hospital a few days before the incident. Since his sister's admission to hospital he felt lonely, tearful, and complained of disturbed sleep. He admitted to the reported incident but would not explain it. As it appeared that he had had an aura preceding it and it was felt he should be investigated for a possible organic lesion, he was admitted to hospital. He said he had been living with his sister, with whom he had an 'odd' relationship, for over 60 years. He apparently used to 'regress' to babyhood from time to time and his sister would then bottle-feed him or give him baby food, and dress him in nappies and take him out in a pram. These periodic regressions occurred from an early age, roughly once every six months, and were confirmed by his sister when she was alive. During some episodes, 'nannies' would come and care for him. He maintained that these episodes were preceded by a forewarning which he could not describe. He said the episodes had not changed in intensity or frequency over the last 50 years.

He suggested that the onset of this disorder was when he was six years old, following an injury when he was kicked in the head by a horse; he was in coma for six months. Hospital records detailing the exact nature of the injuries and the extent of brain damage were not available. When he recovered, he markedly regressed, and his mother was advised by the local general practitioners that she could do nothing but nurse him like a baby. This she continued to do for the rest of her life. He did relearn walking and talking, and between the ages of 11 and 14 years he attended an infant school, but he never progressed further.

His working life had been limited to a little gardening for his father's employers.

Various investigations, including skull X-ray, were all normal. The psychometric tests (verbal IQ of 105, performance IQ of 91, and full-scale IQ of 99) were not suggestive of brain damage. However, the electroencephalogram (EEG) was reported as "an abnormal recording of generalised disturbance not particularly in the temporal lobes and not showing specific epileptic features". It was thought at the time that he probably had an underlying depressive illness and he was treated accordingly.

On discharge, he regularly attended the out-patient clinic and he was also visited at home, where his baby equipment was seen. This included an adult-sized cot where he slept, with quilt and pillow of a baby design; many toys, teddy bears and rattles were scattered about. In another room there was a high-chair which was specially made for him and also a custom-made pram which was of adult size. He maintained that he was being looked after by various people whom he called 'nannies', who helped in getting him these items and who came in frequently to get his shopping done and to take him out in his pram. He also had nappies and bibs drying on a washing line, and there was baby food in Heinz containers in the fridge but no other food stuffs.

Correspondence from some of the 'nannies' included statements that he wanted to remain a baby and that no attempt should be made to change him. There was a strong probability that these letters were written by himself.

Over the years, he became physically frail and more infantile. He started to attend the clinic in his night-shirt, with a bib round his neck and a soother in his mouth. As he had problems in washing himself, the district nurse was requested to help him bath, but she refused because of his history. He would not allow a male nurse to handle him. On the other hand, he was socially skilled in some respects and he also fully managed his financial affairs, including payment for services and maintaining his savings account.

With the passage of time, his self-care deteriorated; the house became dirty and occasionally littered with faeces. He was eventually unable to get out by himself. His strong wish to be cared for in his own home, however, were respected. During the last winter he was found to be suffering from hypothermia; he was taken to hospital where he became unconscious and died two days later.

Discussion

The interest generated in this particular case history is its rarity, its extraordinary features, and its persistence until the age of 80. The long history suggests that it could be a fixation rather than a regression. However, exposing himself at his neighbour's doorstep could be considered as paraphilic exhibitionism (Money, 1986). The tokens of the fetish paraphilia are predominantly, although not invariably, inanimate objects and in this patient's case they were cot, high-chair, toys, and dummy.

Some paraphilics have, concurrently with their diagnosis of paraphilia, also a diagnosis of epilepsy. However, the relationship between paraphilia and epilepsy has not yet been fully resolved, although there are a few published cases of paraphilia associated with temporal lobe epilepsy. For example, Mitchell *et al* (1954) reported a man in whom a seizure would be induced if he carried out a recurrent urge to gaze at a safety pin. Immediately after the seizure while still in a confused state, he would then sometimes dress in his wife's clothing. His symptoms disappeared after temporal lobectomy. Again, Ball (1968) described a patient where the paraphilic behaviour emerged with the onset of a temporal lobe tumour. The history given by the patient of the head injury when he was six years old and the fact that he started regressing on recovering from the ensuing coma was corroborated by his sister. Though the EEG did not show definite involvement of the temporal lobe, the recording did show a generalised abnormality. Therefore, it is possible to speculate that the paraphilia originated with the head injury and that it was perpetuated by his mother's over-protective and infantilising attitude. It is interesting to note that two older sisters aged ten and eight years and one brother aged three years died in the same year that he was born from scarlet fever, beriberi, and an accident respectively. This could explain his mother's behaviour, which no doubt influenced his sister's attitude towards him as well. On his mother's demise his sister continued to foster his paraphilic behaviour. This continued until his sister was admitted to hospital, where she eventually died of senile dementia. Dynamically, this patient's paraphilia symbolised a regression to infancy in order to reclaim the attention and love of his mother and to undo his displacement in her affections by his sister's birth.

Two cases of 'diaper fetishism' have been reported. Tuchman & Lachman (1964) reported a 29-year-old man wearing nappies and rubber pants, and Malitz (1966) described a 20-year-old man who had a compulsion to wear nappies in order to defaecate in them; defaecation was always accompanied by orgasm either with or without masturbation. Of the 48 cases of clinical fetishism studied by Chalkey & Powell (1983), there was only one case involving baby clothes. According to Godfrey (1981), regression or regressed behaviour is known to occur in dying patients. Tatro & Marshall (1982) recorded that sometimes older adults may employ regression as a defence mechanism to cope with their foreshortened futures.

Our patient showed periodic infantile behaviour most of his life. The fetishistic nature of his behaviour involved baby clothes and equipment. He also demanded confirmation that he was a baby from authority figures.

Treatment approaches are problematic; some patients come to therapy for symptoms such as depression or impotence. When the paraphilic behaviour is discovered by the family, the patient is sometimes encouraged or coerced to seek treatment – for instance a paraphilic transvestite's wife may insist on her husband seeking help as a condition of saving the marriage. This patient first came to notice because of the incident of indecent exposure and he was initially treated for a presumed underlying depressive disorder. Over the years, however, there was little evidence of affective illness.

Aversive behaviour therapy poses ethical problems in attempting to treat a behaviour that a patient views as enjoyable and does not usually harm others. It can be argued that such behaviour limits the individual's ability to relate fully to others. If the paraphilic does not voluntarily seek treatment, the therapist is at great disadvantage in forming a treatment alliance. In this case the patient did not wish to change his behaviour and his wishes were respected.

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Kleptomania in a 13-Year-Old Boy

A Sequel of a 'Lethargic' Encephalitic/Depressive Process?

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A 13-year-old boy developed severe kleptomania after a depressive illness. This was a response to psychological trauma, and appeared in the context of a probable 'lethargic' encephalitic process.

Kleptomania is mentioned as a diagnosis in current classificatory systems in psychiatry, but little is known about it and the literature in the area is jejune. The person repeatedly fails to resist impulses to steal objects, which are not acquired for personal use or monetary gain. They may be discarded, given away or hoarded. The central psychopathological mechanism appears to be loss of impulse control but in the DSM–III differential diagnosis (American Psychiatric Association, 1980), pathological stealing should be distinguished from: recurrent stealing by healthy people (acts are planned and there is an obvious motive of personal gain); stealing which is part of a conduct disorder or antisocial personality disorder; organic mental disorder with memory impairment and intellectual deterioration; psychotic states. The condition is very rare in childhood.

Case report

John, aged 13 years, was transferred to our regional child psychiatric unit from a paediatric ward. Encephalopathy, presumed but not proven to be of viral aetiology, had been diagnosed, and he was profoundly depressed.

His symptoms had started eight months before admission (in December) with a flu-like illness followed by weight loss. We have few details of this illness. Symptoms included myalgia, lethargy, anorexia, mild disorder of the upper respiratory tract, and mild pyrexia. It appears that the majority of these symptoms resolved but were followed by marked weight loss. Five months afterwards his grandmother, who had been the primary carer for John since he was two years of age, attended the funeral of one of her daughters abroad. During this time John discovered the tragic details of the death of his mother, who had been brutally stabbed to death together with her four-year-old son when John was two years old, and he found out that, contrary to his belief until then, his grandmother was not his mother. When his grandmother returned she found John would cling to her and be overanxious of her well-being. Three months after her return John was admitted to a paediatric ward with 19 kg weight loss (three stone) in seven months, and two-week history of night sweats, listlessness, lethargy, withdrawal and anorexia.

On admission to the paediatric ward he had a low-grade pyrexia with no other signs. However, he became increasingly lethargic and miserable. He was noted at times to fall into a deep sleep during the day and he seemed very confused on arousal. He became more and more drowsy, sleeping for most of the day and at times he was barely rousable. His depression deepened and he would weep uncontrollably. These catastrophic reactions were precipitated by talking about his mother's death.

Investigations included two electroencephalograms (EEGs) (performed within one week during the acute 'lethargic' phase of illness), which were grossly abnormal, with diffuse slow-wave activity, suggestive of diffuse encephalopathy and encephalitis. A computerised tomography (CT) scan and laboratory and clinical tests proved normal. EEGs performed two to three weeks later showed an improvement.

On admission to the psychiatric unit, physical and neurological examinations were normal. He wanted to spend his time sleeping in a foetal position, refused to do anything for himself, and shouted in a loud monotonous voice 'my legs', 'help me'. He complained of 'stabbing pains' in his legs which stopped him from standing unsupported although he was physically able to do so. He often described his behaviour as being that of a two-year-old and would only eat mashed, wet food. He looked profoundly depressed and would slap his face, shouting at nurses 'kill me', 'finish me off'. He talked frequently of frightening dreams and of not knowing the difference between dream and reality. He was reluctant to talk about his 'real Mum'.

John was the third youngest of four siblings. All children were in the care of the local authority and had been brought up by the maternal grandmother, the youngest two believing her to be their mother. The grandmother had cared for the children adequately, if anything tending to overindulge John. The eldest son had a history of minor behavioural problems. John's birth and early development were entirely normal. Before his illness he had had minor behavioural problems and was described as slightly below average ability in school. He was described as disruptive and immature at school, though likeable and sociable. There had been occasional truanting and one episode of shop-lifting with friends.

There was a strong family history of psychiatric illness on the paternal side of the family. Details are lacking since our only informant was John's grandmother and we were unable to obtain psychiatric records. John's natural father was admitted to psychiatric hospital following his wife's murder, for treatment of depression. Several of his siblings had a psychiatric history and the youngest sister, who was an accomplice to the murder of John's mother, was found to be suffering from morbid jealousy and was committed for treatment. A few months before John's admission to our unit, his grandmother's only remaining daughter was murdered abroad. We do not know who murdered either John's mother or aunt, but do not believe the two events to be connected.

Our formulation was that in the context of a probable viral encephalitis and in response to psychological trauma, John had developed a severe depressive illness. He was treated with much encouragement and intensive physiotherapy as a means of restoring function and activity and reducing withdrawal. He had individual therapy sessions and we conducted joint sessions with him and his grandmother for facilitating talk about his mother's death and grieving by both.

He was also started on imipramine, increasing to 100 mg daily. After two weeks he became less sleepy, more alert and mobile, and he no longer complained of pains in his legs. He still, however, expressed a wish to be dead. Concurrent with his leg pains resolving, he began stealing (money, purses, pens and jewellery were disappearing from offices, drawers and lockers). He was devious. When discovered he became distressed, hit himself, and would shout "kill me, I wish I were dead". He promised faithfully not to steal again but within minutes he would be looking for opportunities. He did not spend the money or make use of any of the items, but simply hid them in his bed. Behavioural therapy failed. When the stealing started he said that he did it because when he was found out we would kill him. As his mood improved he ceased to use this explanation but could not give any other.

At this time we were able to complete psychometric testing. His full-scale IQ was 68 (77 on the verbal scale, 63 on the performance scale), and his reading age was 9 years 11 months. These results were likely to be an underestimate, as he was lethargic and drowsy during testing. When his reading age was checked three months later (on a different test), it was 11 years 1 month, with a comprehension of 11.8 years.

Repeat EEGs (six weeks and four months after the initial abnormal recordings) showed a low-voltage desynchronised record, poorly formed for the patient's age but otherwise normal. A repeat CT scan showed the ventricular system to be a little full but not grossly abnormal. The cortical sulci were also large, but no focal lesion was seen. There were no areas of altered attenuation or pathological enhancement.

Within two months of starting his antidepressant medication he was considered to have recovered from his depression. However, as he emerged from this he showed many features of a personality deficit: disinhibition, impulsiveness emotional lability, and lack of affective depth. Emotional lability was observed particularly with respect to his compulsive stealing: when confronted with it he would become distressed, shouting that he wanted to be dead and weeping, but then within seconds he would be laughing and joking again. When confronted at later stages in his condition he would become aggressive and threatening. He was talkative but repetitious in his topic of conversation. He showed little interest in ward and school activities and he remained prone to sleep during daily activities.

His stealing became increasingly uncontrollable and extended well beyond the confines of hospital and home. He was picked up and detained by the police on numerous occasions and had several court appearances, but his stealing continued unabated. Containment and placement became a major problem, with neither his family nor psychiatric or social services being able to provide an adequate setting for control and treatment. Ten months after he was first admitted to the psychiatric unit he was eventually placed in a secure local-authority unit for delinquent boys. The monitoring of his psychiatric state was taken up by the forensic adolescent psychiatric services. He remained at the secure unit for nearly one year with little change in his condition and no recurrence of a depressive state. He was then transferred to the behavioural management unit for psychiatrically disturbed adolescents of a private psychiatric hospital.

Discussion

This boy's stealing was characteristic of kleptomania as described in DSM–III. The thefts were recurrent, impulsive and apparently senseless, and John was observed to become tense before the act and to derive pleasure from it. The stealing started during his recovery from a severe depressive state and in the initial stages it was linked to his morbid thought content. Its persistence, however, after the affective state had resolved can be explained in terms of the remaining personality changes which accord best with DSM–III's organic personality disorder, characterised by emotional lability, impairment in impulse control, apathy, and indifference to most activities. In view of the previous profound depression, we considered the possibility of mania, but there was no clear evidence of an elevated, expansive or consistently irritable mood or of excitement, flight of ideas, or convincing grandiose ideas, and the stealing continued after his antidepressants were stopped.

Excessive sleepiness and lethargy were features of the condition. Although these symptoms can occur in depressive states, the associated encephalopathic signs and the personality sequelae raise the issue of whether this was an abortive form of encephalitis lethargica. Many authorities consider that encephalitis lethargica disappeared completely before the onset of World War II. However, possible cases have been reported since (Leigh, 1946; Espir & Spalding, 1956; Rail & Scholz Swash, 1981). Moreover, polymorphism was a striking feature of the disease (epidemics differed both in acute phases and in incidence of sequelae) and the possibility has been raised of present-day mild or attenuated clinical pictures – particularly in children and young adolescents – increasingly dominated by psychiatric manifestations and resulting in considerable subsequent psychiatric disability (Lishman, 1987). Our patient showed several features described by von Economo in his classic account in 1929: peak winter incidence, a pre-clinical phase resembling influenza, hypersomnolence persisting for several weeks, rapid debility, weight loss, and low-grade pyrexia. Investigations performed during the acute phase failed to isolate a causative agent. Neurological signs were lacking in this case; however, these, in particular disturbance of ocular movements, may be fleeting and it is possible that they were missed.

The account by Lishman (1987) of a typical post-encephalitic personality could certainly be directly applied to this patient. Fairweather (1947) gives a vivid illustration of severe sequelae in post-encephalitic patients without Parkinsonism or gross physical defect admitted to Rampton State Institution for violent and dangerous patients. These included damage to property and theft, as well as damage to others, sex offences and self-injury.

Our case highlights the lack of adequate services for adolescents with problems such as this boy's and raises the issue of what type of facilities should be available for the treatment/containment of adolescent recidivists with probable post-encephalitic personality disorders.

Severe psychopathology such as this boy's is rare in childhood but it can lead to considerable psychiatric disability. This case suggests that severe or particularly strong psychological stresses as well as biological factors may be required for its emergence.

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Contents: Insight and psychosis/The 'new cross-cultural psychiatry'/Progesterone prophylaxis?/Benzodiazepine withdrawal/ECT in neuroleptic malignant syndrome/Was Hitler a Christian?/The musical idiot savant/Anorexia nervosa and OCD/Toad-lickers psychosis – a warning/Self-catering during rehabilitation/A Freudian lacuna?/Genetic origins of psychosis/Post-traumatic stress disorder/Malaria presenting as atypical depression/Anorexia nervosa in people of Asian extraction/Temporal lobe in schizophrenia

Insight and psychosis

SIR: David's comments on 'Insight and psychosis' (*Journal*, June 1990, **156**, 798–808), and those of other authors quoted by him, that "a surprising proportion of patients do possess insight", fully confirm my own experience.

In an essentially community-orientated adult psychiatric service, clinicians such as myself rely more than has traditionally been the case on patients assuming a considerable degree of responsibility for their illness and its treatment, and indeed for their general behaviour in a public context. It has been necessary, and increasingly a delightful surprise, to discuss fully with those we call 'long-term mentally ill' what Dr David has called their insight.

I have found that patients' comments are often at first misleading. One man in his fifties recently told me that he was definitely not mentally ill and did not require medication. This was our first meeting and he mistrusted me. Later, however, it transpired that he knew very well he had been suffering symptoms of schizophrenia much earlier in life and that depot neuroleptic medication controlled these symptoms more or less completely. He was anxious, however, that this admission would result in the removal of his Home Office Warrant, and that he would thereby lose his benefits and be forced to seek employment.

I was able to reassure this man that he would continue to receive benefits and could continue to attend the industrial workshop as before. The example demonstrates a further advantage for the clinician in exploring fully with the patient his or her insight. The therapeutic relationship deepens. Above all I have found in a number of cases that a patient's self-esteem rises spontaneously and almost palpably when their own grasp of their illness, often astonishingly sophisticated, is given professional credence.

May I therefore endorse Dr David's conclusions. May I suggest that psychiatrists do not wait for the results of formal research, and that they undertake informal research of their own with the very next long-term mentally ill patient they encounter. Too convincingly, I believe, have we been trained to attempt the imposition of our expectations on our patients, and we do so without recognising the burden, the awkwardness, the mistrust it places upon ourselves. Letting patients speak for themselves and take responsibility for themselves is a very liberating experience.

Finally, I wonder that Dr David omitted from his Appendix questions designed to clarify whether insight into another's illness might be preserved despite the loss of personal insight. Perhaps the publication in preparation (*The Assessment of Insight* by A. S. David & G. Nestadt) will put this right.

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SIR: David's thought-provoking article (*Journal*, June 1990, **156**, 798–808) seems to raise as many questions as it answers. My unease about it is due to the extent to which he assumes observer uniformity when assessing psychotic patients. He limits his use of the term 'insight' to the subject's appraisal of his mental state and his ability to label certain mental phenomena as morbid – "it simply requires the acceptance of personal illness affecting the mental apparatus". This seems fine at face value and even if the term 'illness' is dropped and 'change' used it still ignores the observer.

Alas, psychiatrists working in everyday practice are not as objective as we would like to think. Kreitman *et al* (1961) has shown that inter-rater reliability between observers for positive and easily defined symptoms is low. While standardised interviews do improve inter-rater reliability, the vast majority of ordinary psychiatric assessment is done on a more *ad hoc* basis and clearly insight is open to misinterpretation in this setting.

Secondly, the concept of pseudo-insight seems an important one. The hermeneutic value of an intellectual explanation of mental illness is important, but the form of an individual's appraisal of his mental disorder seems more significant than the content. Accepting treatment is one aspect of this, but perhaps an allowance needs to be made for the manner in which acknowledgement of medical illness comes about. There is a world of difference between the patient who says "I must be mad because you say so" and the resigned statement "you're right doctor, I'm breaking down again".

A third aspect relates to the psychiatrist's knowledge of mental disorder. His or her knowledge is generally by description (as opposed to by acquaintance). As insight is ultimately a clinical judgement of a patient by a doctor, what happens is for descriptive knowledge to be used to assess an experience that is classified as knowledge by acquaintance. There may not be a problem in this regard, but if knowledge by acquaintance is the route to insight, there seem no grounds on which to contradict a patient who tells his doctor "I have insight" when in reality he does not.

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KREITMAN, N., SAINSBURY, P., MORISSEY, J., *et al* (1961) The reliability of psychiatric assessment: an analysis. *Journal of Mental Science*, **107**, 887–908.

SIR: We agree with David's contention (*Journal*, June 1990, **156**, 798–808) that insight is best regarded as a multi-dimensional phenomenon. We would support Dr David's view that one such dimension is the ability of the patient to 'relabel' unusual mental events as pathological. However, we take the view that compliance with treatment should be seen not as a dimension of insight, but rather as a related phenomenon – as Lin *et al* (1979) have demonstrated, the correlation between insight and compliance is limited. This suggests that the schedule proposed by Dr David, which allows compliance itself to carry considerable weight, overemphasises the contribution of this variable to the core phenomenon.

The mechanisms underlying diminution of insight remain obscure, but are receiving increased attention. Insightlessness may be regarded as: (a) a normal phenomenon, insofar as many people demonstrate limited awareness of certain characteristics of their personality and behaviour; (b) a defence mechanism (denial); (c) a delusional phenomenon; (d) a feature of the schizophrenic defect state; and (e) a specific defect of cognition.

We have been attempting to operationalise the concept of insightlessness in schizophrenic patients. Given that direct measurement of the components of insightlessness is not possible, our proposed scale attempts to derive an overall measure, based on a semistructured interview. The scale distinguishes between attitudes to overall management and those to compliance with physical methods of treatment. Additionally, the scale permits measurement of behaviour in response to changes in psychopathology, perhaps the most important indicator of insight. An assessment of attitude to previous episodes of illness is included, an element which varies considerably between patients. Allowance is made for those subjects who reject the philosophical concept of mental illness, since it would be inappropriate to necessarily regard those as insightless. The scale measures insightlessness rather than insight, since the former has greater and more relevant clinical utility. (The schedule and score sheet are available from the authors.)

A pilot study of 13 patients fulfilling diagnostic criteria for schizophrenia indicates that scores derived from use of the schedule correlate well with global clinical impressions of insightlessness. In many of these patients, the degree of insight was not obviously correlated with the extent of delusional conviction.

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AUTHOR'S REPLY: I am delighted that Dr Culliford finds my discussion on insight in accord with his clinical practice. The point about one patient's insight into another's delusions is a fascinating one as it suggests that the basis mechanisms of logical inference leading to a delusional misinterpretation may be domain-specific rather than generally impaired. This is analogous to agnosias confined to certain classes of things seen in the neuropsychological literature (e.g. Warrington & Shallice, 1984). Dr Wear chooses to tackle the vexed issue of assessing insight. He is a little unfair in invoking Kreitman's classic 1961 paper, as our profession has moved on since then. The World Health Organization in the *International Pilot Study of Schizophrenia* (WHO, 1973) found that insight, defined operationally, achieved respectable inter-rater reliability coefficients of around 0.77. However, the deeper question is how we interpret the attitude behind the words of our patients. Here we begin to lose on the swings of reliability hopefully to gain on the roundabouts of validity. When it comes to treatment compliance it seems most safe to consider both what the patient says and what he or she does.

The schedule proposed by Drs Lambert & Baldwin has much in common with my own. However, they are mistaken in their attempts to pin down the 'core phenomenon' since I propose that there is no single core but at least three separate but overlapping constructs. It is not surprising that 'insightlessness' as measured by their scale does not correlate with delusional conviction since this aspect of insight is ignored. Patients may have convictions, perhaps of delusional intensity, as to whether they are ill or not but this is separate from other delusions concerning their bodies, minds or the state of the world. Exploring these relationships will teach us much about the nature of psychosis.

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The 'new cross-cultural psychiatry'

SIR: The invitation to contribute a review article on recent developments in psychiatry and anthropology was welcome. When my article was published (*Journal*, March 1990, **156**, 308–327) I was somewhat disconcerted to find it appearing in an issue specially devoted to 'cross-cultural psychiatry'. Disconcerted, because I had suggested that current work in the two disciplines argued that contemporary psychiatry could be faulted for ignoring the context of its own assumptions and methods through relegating the cultural domain into something called 'transcultural psychiatry', whose subject matter was that of ethnic minorities and non-British and non-American communities. Precisely what the other papers were about.

My surprise was compounded by the unprecedented editorial by one of the associate editors (Leff, *Journal*, March 1990, **156**, 308–307) which did not attempt to introduce the papers, or indeed 'cross-cultural psychiatry' however defined, but instead was concerned solely with my paper. While one should doubtless be flattered at being singled out for the sort of 'health warning' it offered, this novel procedure does raise certain questions about the editorial impartiality of the *Journal*. Surely the place for scholarly debate and criticism is the space devoted to your correspondence columns, not a preceding editorial?

In this editorial, Professor Leff makes certain flippant assertions about the newer approaches, which he derives from my review but which remain by and large mistaken. His proposals for further work are generally unexceptional and recapitulate sections of my paper, but he simplifies the notion of a biological-sociological explanatory continuum which I point out is a conventional representation, not the basis for seriously considering the relative contribution between the biological and the social, a basis which is impossible when we are concerned with a dialectical relationship in which each responds to the other in a complex manner (Simons, 1985).

In one respect Professor Leff reiterates a conventional error. Culture is not cultural distance even if the latter is more easily measured. In attempting to ascertain the cultural contribution to psychopathology he suggests the problem is simplified if we hold culture constant, as in his comparison of patients from Salford and London. The baby has however followed the bath water (to employ his aphorism) for, if culture is held constant in a study of difference, then the only observable differences which remain are these of individual, biological and psychological variation. To take a rather simpler instance, if access to nutrition is in part determined by class status we find associations between cultural position and physical height: in an egalitarian society culture determines nutrition rather less and thus differences in height are determined especially by hereditary factors. That does not make 'height' *per se* a genetic phenomenon. Similarly, if we reduce cultural psychiatry to comparative epidemiological studies in which we attempt to control for culture, culture vanishes to make the phenomena of interest apparently biological or psychological in nature. It then has to be reintroduced through focusing, as Professor Leff puts it, on "the societies that are most culturally different from the west, and hence of greater interest". If all societies are becoming increasingly similar, as he argues, then patterns of, say, overdoses presumably become similar and one eventually concludes that overdoses in general are not a social phenomenon. Not surprisingly, two papers in that issue of the *Journal,* both concerned with the perceived consequences of parasuicide, were not in the 'cultural' section.

The newer critiques do not, as Leff suggests, necessarily fault psychiatry's *aspirations* to be a scientific discipline: that is one that seeks explanations independent of the observer's perspective. That at least should be clear from my review. Indeed I myself warn against the too casual neglect of evidence from the biological sciences. The problem is that the wish to be scientific, and the claim to be scientific, are often very different from actually being so. The difficulty with much of the older transcultural psychiatry, carried out by psychiatrists untrained in any social science, is that it mistakes the particular for the universal, the contingent for the necessary, the political for the biological.

To distinguish the two sets of categories is presumably the aim of any attempt at scientific truth. In certain particulars we find that the context of observation determines the observed events to the extent that locating patterns such as overdoses or agoraphobia solely in a person's individual characteristics does not help us to interpret the pattern at all.

These difficulties are not of cause restricted to psychiatry. My speculation as to the value of 'pathology' (or disease) is not, as Leff protests, some sort of Laingian romanticism but a concern as to whether any conception of 'pathology' is truly useful in scientific terms: we are, as the medical student joke has it, on the side of the human not the virus. Fair enough, as humans embedded in our illnesses and in our struggles we are compelled to act, but the prescriptive urge is not necessarily the appropriate ground for understanding. My suggestion in the paper which Leff refers to (Littlewood, 1984) that the experiences associated with what we psychiatrists conventionally refer to as cerebral pathology may at times be taken up by societies in certain situations as meaningful experiences is not an exhortation to consider this as the real meaning, merely that it can occur and that it may have interesting theoretical implications for us. I deal with this possibility at greater length in a forthcoming volume which uses field data including conventional Present State Examination assessments (Littlewood, 1990).

Professor Leff's editorial, its appeal to the demands of the 'practical difficulty' as justification for the validity of the findings, is an instance of the confusion between fact and value which many of us associated with the 'new cross-cultural psychiatry' argue is inevitable. We do not however pretend it can always be avoided, or allow value to masquerade as fact. A little closer examination of epistemology, of actually learning to distinguish the baby from the bathwater, will not come amiss.

Incidentally, in his put down of local knowledge, Professor Leff characterises the Yoruba masculine power Shopana (Shopona, Sopono) as a 'goddess'. I am not sure if he is thinking of the probably cognate Ewe/Fon power Shapata which is sometimes represented as a generic or androgynous emanation of the Mawu/Lisa principle. To characterise these concepts as free-standing deities rather than powers, principles, faculties or even mechanisms is, in any case, problematic. This is not trivial schōlasticism but an example of the sort of problem we run into when we interpret others' meanings through our own frameworks. Our own categories of neurosis are hardly independent of assumptions about gender.

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Progesterone prophylaxis?

SIR: I was most surprised to read Meakin & Brockington's statement that "Progesterone is widely used in the treatment and prophylaxis of post-natal depression (*Journal,* June 1990, **156,** 910). To my knowledge, progesterone has never been used successfully as a treatment for post-natal depression. In fact, prolonged administration of progestogens may lead to depressive symptoms (Silverstone & Turner, 1982). Dalton (1985) claimed that progesterone prophylaxis was successful in reducing a recurrence rate of post-natal depression from 68% to 10%. However, the study was flawed in two ways. Firstly, it was not double-blind. Also, there was no attempt to standardise the diagnosis of post-natal depression – cases were divided into mild, moderate and severe pending on how they were treated by their various general practitioners.

The fact that the authors were encouraged to use progesterone as a sole therapy for puerperal mania by their experiences with two patients quite frankly astounds me. The first patient reported a 'subjective calming effect' when progesterone was given (50 mg intramuscularly) before and after neuroleptic therapy was commenced – nothing particularly encouraging about that. The second women's improvement was most likely due to the fact that she was given haloperidol (40 mg intramuscularly – a high dosage) and chlorpromazine (50 mg intramuscularly) during the 48 hours before recovery.

Clearly, hormonal changes in the puerperium may be one of the factors that precipitate a psychotic illness in susceptible individuals but to expect that progesterone might be successful as a therapy for puerperal mania is in my view being rather simplistic.

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Benzodiazepine withdrawal

SIR: With reference to Ashton *et al*'s paper on buspirone in diazepam withdrawal (*Journal*, August 1990, **157**, 232–238) we would like to make the following points.

We question the clinical relevance of this study. It is now generally accepted that gradual dose reduction with attention to appropriate psychological treatment is the best way to manage benzodiazepine withdrawal (Edwards *et al*, 1990). If this is done at the patient's own rate, pharmacological treatment of symptoms, which may complicate the withdrawal process, as indeed occurred in this study, should be unnecessary.

We think the study was unethical for two reasons: firstly, withdrawal was rapid and took no account of either the starting dose or the patient's response to withdrawal; and secondly, a blind study deprives the patients of the right to determine their own rate of withdrawal and the opportunity to learn from this experience. Did informed consent include telling patients that this was not the best way to come off benzodiazepines, was likely to create unnecessary distress and that buspirone was unlikely to help?

The design of the study is unsuited to small numbers. Unmatched groups, a failure to control for attending a support group or concurrent prescribing, and the high drop-out rate in the buspirone group make it difficult to draw any useful conclusions.

Finally, the study ignores important psychological factors which are crucial to maintaining abstinence. The importance of patients being in control of their withdrawal, learning non-drug alternatives and improving their quality of life makes a psychological approach more appropriate than a pharmacological one.

This study only perpetuates the search down a blind alley for pharmacological short-cuts which fail to respect the patient's right to participate in the decisions, manage the withdrawal and be offered alternative ways of coping.

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EDWARDS, J. G., CANTOPHER, T. & OLIVIERI, S (1990) Benzodiazepine dependence and the problems of withdrawal *Postgraduate Medical Journal*, **66** (suppl. 2), S27–S35.

SIR: I would like to congratulate Cantofer *et al* (*Journal*, March 1990, **156**, 406–411) on their study. Benzodiazepine dependence is a difficult condition to treat and an attrition rate as low as they obtained must indicate considerable enthusiasm. However, I am slightly surprised at the design of the study which appears confounded by having two variables, in that patients were allocated either to abrupt withdrawal and active treatment with propranolol or gradual withdrawal and placebo propranolol. From the study design one could draw the spurious conclusion that propranolol is no benefit for the patient withdrawing from benzodiazepines. I believe there is fairly good evidence that propranolol is of benefit in benzodiazepine withdrawal, at least as far as somatic symptoms are concerned (Halstrom *et al*, 1988). The other main finding of the study, that gradual withdrawal is easier than abrupt withdrawal, is already well supported in existing literature. However, to make such a deduction from the study is an error in logic because of the confounding variable of active or inactive treatment.

Perhaps I have overlooked some strength in the methodology chosen; I would be glad to be corrected if this is the case.

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ECT in neuroleptic malignant syndrome

Sir: In his otherwise impressive update on neuroleptic malignant syndrome (NMS), Kellam (*Journal*, August 1990, **157**, 169–173) seems to have included some factual inaccuracies while discussing the treatment of continuing or recurring psychosis after the successful treatment of NMS. Dr Kellam quotes Lazarus (1986) to the effect that electroconvulsive therapy (ECT) is safe after the syndrome has subsided. Lazarus, in his case report, suggested that ECT may offer a safe and rapidly effective intervention in cases of NMS unresponsive to supportive medical therapy.

Similarly, Hermesh *et al* (1987) recommended ECT for the treatment of NMS episodes rather than for continuing or recurring psychosis after the successful treatment of NMS. In my rejoinder to Hermesh *et al* (1987), I did not argue about the safety of further neuroleptic treatment for psychotic illness as Kellam seems to imply. I suggested that the drug treatment (i.e. bromocriptine or dantrolene) should be tried for the management of NMS before resorting to ECT (Adityanjee, 1987). I did mention having used ECT for the management of recurring psychosis after NMS had subsided (Adityanjee, 1987; Adityanjee & Chawla, 1989). Similar practice was adopted by Aizenburg *et al* (1985) who used ECT for the management of psychotic illness after NMS had resolved successfully with supportive treatment only.

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Was Hitler a Christian?

Sir: There are a number of dubious assumptions and implications in Samuel's (*Journal*, July 1990, **157**, 151) argument that Hitler, Mussolini, Stalin, Franco and Hoess were all Christians. Firstly, there is the assumption that anyone who professes to be a Christian is one by definition. Arguably, such a profession does fit the dictionary definition given by Philip Timms, although I would feel that in this context the use of the word 'profess' should more meaningfully be taken to include observance and practice of, rather than simply confession of, faith. Secondly, there is an implication that the failure of the Roman Catholic Church to proscribe *Mein Kampf* amounts to an acceptance of its contents not only by the Catholic Church (which is itself highly debatable) but also by the wider Christian Church as a whole.

The most serious implication of Dr Samuel's argument, which is both unjustified and offensive, is that there is some causal connection between the professed Christian faith of the individuals named and the acts of atrocity and inhumanity that they perpetrated during their lives. The lives that they led, as recorded in history, clearly betray the insincerity of any profession of Christian faith that they made. The teachings of Christ made clear that it was not those who 'professed' righteousness, but those who were truly repentant, who found favour in God's sight.

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This correspondence is now concluded.

Anorexia nervosa and OCD

Sir: I read with interest the paper by Holden (*Journal*, July 1990, **157**, 1–5) on the evidence for a relationship between anorexia nervosa (AN) and obsessive–compulsive disorder (OCD). However, I find his analysis too selective and would like to suggest several missing points.

(a) Evidence suggestive of a positive relationship

(i) Abnormal brain serotonin has been reported in both AN and OCD. Thus tryptophan has been used to treat both conditions, while a recent open trial of fluoxetine, a serotonergic reuptake blocker, has shown favourable result in weight maintenance of AN patients (Weltzin *et al*, 1990). This trial was based on the belief that AN is related to OCD, which responds to fluoxetine treatment. This weakens Holden's conclusion that there is currently little advantage in joining the two conditions "until linkage of the syndromes is shown to have meaningful benefits for management".

(ii) Maudsley Multiphasic Personality Inventory profiles of AN and OCD patients are remarkably similar, both showing abnormal elevations on the scales of depression, psychopathic deviate, psychasthenia and schizophrenia (Goodwin & Andersen, 1984; Carey *et al*, 1986).

(b) Evidence against a positive relationship

(i) Psychodynamically, AN is a defence against underlying deficits in the sense of self, identity and autonomy, which is believed to be due to a paucity of encouraging responses to child-initiated cues in early parent–child interactions. The characteristic defence mechanism of the AN patient is rigid denial. In contrast, OCD is usually construed as a regressive fixation at the anal-sadistic era of psychosexual development, which is characterised by the defences of undoing, isolation, displacement and reaction formation.

(ii) In terms of psychological treatment, behavioural therapy alone is generally ineffective in AN patients who typically defy treatment contracts and create power struggles, but is the most important form of therapy for OCD. If AN were an authentic "compulsion neurosis" as noted by the author, it should respond well to exposure and response prevention. Besides, dynamically oriented individual therapy is the cornerstone to the management of AN, but is often pessimistically contra-indicated in OCD.

(iii) Although both conditions are regarded as multifactorial in aetiology, current evidence is generally moving more towards sociocultural hypotheses for AN, but neurobiological for OCD. This is hardly examined by the author.

Overall, there is suggestive neurochemical and psychometric evidence, debatable phenomenological resemblance, tenuous genetic and epidemiological data, and contradictory treatment results to support AN as an OCD. Theoretical arguments apart, anyone who has worked in psychotherapy with these two groups of patients will not dispute the striking dissimilarity in the therapist's reaction towards the resistant, unconcerned ("let me be") anorectic patient, and the distressed, eagerly help-seeking ("let me not be") obsessive–compulsive patient. For this reason alone, I believe they are disparate conditions!.

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Toad-lickers psychosis – a warning

SIR: Since we in England generally follow our American colleagues in experiencing novel epidemics of psychoactive substance abuse, the following warning may be timely. The skin glands of bufo toad species secrete the hallucinogen bufoterine, which although having only 0.1% of the hallucinogenic potency of lysergide, is active orally and produces the same effects. The Australian cane toad is popularly kept as a pet in the US, and licked by its owners for the resulting hallucinatory effects. The phenomena has caused so much concern that the state of South Carolina is considering legislation to ban ownership of cane toads. Bufoterine is a monoamine oxidase inhibitor, blocks uptake of noradrenaline and has complex effects on reflex activity in the spinal cord (Bowman & Rand, 1980). These active properties are responsible for the more unpleasant side-effects of toad-licking: sweating, distressing palpitations, vomiting and involuntary defaecation.

The two native English species of toad, the common toad (*bufo bufo bufo*) and the natterjack toad (*bufo calamita*) share the cane toad's hallucinatory potential. There are as yet no reports of toad-licking in the UK, and we would be very interested to hear from any psychiatrists whose patients admit to this bizarre form of drug abuse.

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Self-catering during rehabilitation

SIR: Self-catering is an important element of rehabilitation. We investigated the food purchases of a self-catering ward for rehabilitation of drug abusers (80–90% former opiate addicts), located in Springfield Hospital in south-west London. Establishing good dietary habits may aid recovery from the accumulated effects of chaotic or debilitating life-styles, and may also help to sustain a drug-free state. A good nutritional state can be an indicator of successful treatment of dependence (Kleber, 1989).

The initial request for the study came from the patient community meetings. During four weeks in Autumn 1989, all self-catering patients cooperated in a survey of their expenditure on food and non-food items, using an itemised food diary. Data on 126 patient-days were collected. Nutritional analysis of these food items was performed, using the DIET 2000 programme, for each week. Results were compared with the recommended daily amounts (RDA) for nutrients (Department of Health & Social Security, 1979) and the recommendations of the National Advisory Committee on Nutrition Education (NACNE, 1983). Food expenditure was compared with values from the latest Ministry of Agriculture, Fisheries & Food (MAFF, 1989) national food survey. Of course, purchases do not equal intake because of wastage and nutrient loss in preparation, and it was necessary to make a (generous) estimate of milk consumption – 1 pint/patient-day.

Overall, the patients purchased food with high energy values (maximum week = 4336 kCals, 18.23 MJ), reflecting excessive amounts of sugar and fats and disproportionately low contributions of protein and starch. Absolute amounts of vitamins, minerals and fibre were often very low. Such purchases are typical of the clinical picture of the 'obese malnourished' frequently encountered in clinical dietetics.

For example, the percentage of energy derived from sugar peaked at 30.1% total kCals (NACNE recommendation = 12%). Fat contributed up to 51.6% of the total energy (NACNE recommendation = 34%, and the average for south-east England is 40%), and protein contributed as little as 7.5% (NACNE recommendation = 11%). Nutrient balance was worst during a week when only three male patients were shopping. At that time, amounts below the RDA were purchased of protein, fibre, iron and certain vitamins (thiamine, riboflavin, B6, C). Very low amounts of zinc (5.6 mg) and folate (98.5 µg) were bought (compared with the US National Academy of Sciences' RDAs). Three kilograms of white sugar were purchased during this week, and frozen chips and baked beans were the only vegetables. No fresh fruit was purchased during the whole four weeks.

The food budget was funded by the hospital catering department on the basis of in-patient food allowances: £12–£18 was the hospital's weekly patient food cost during the study period. Such hospital food costs are based on large-scale contract purchasing at lower prices than retail supplies. The specific purpose of this budget was not clear to the patients, and only £10–£15 was used for food purchases. This was well below the 1988 National Food Survey average values for food (£17.01 for adults, all sources) and below the 1988 values for the poorest 'E2' non-earner sector of society (£15–£36, all sources). Food expenditure is higher in south-east England than the national averages, and figures for inflation in food costs for 1989 were about 7% (unpublished, MAFF). A total of £12–£18 is thus considerably less than the likely food costs of even the poorest section of the local community at that time.

It seems imprudent to set convalescent patients a task at which they are bound to fail. Unrealistic resourcing, inadequate shopping skills, poor knowledge of meal planning and limited cooking skills combined to produce a poor diet and unhealthy eating patterns in these self-catering patients. On completion of this study, the researchers involved presented the findings to the patients. Subsequently the ward gained a full-time occupational therapist who, together with the ward nurses, incorporated budgeting, shopping and cooking into the therapeutic programme. The food budget was reviewed by the managers and additional funds are now available from a non-catering source, given that additional funds were not available from the catering budget in the financial year.

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A Freudian lacuna?

SIR: In his article on the psychoanalytical aspects of morbid jealousy in women (*Journal*, January 1990, **156**, 68–72), Freeman seems to be saying that "the phallic sexual organisation" of his two neurotic female patients was instrumental in their downfall (figuratively speaking). Does he mean that possession of such an apparatus would therefore also be aetiologically significant in the development of morbid jealousy in male patients, since they are ordinarily so endowed by nature? Or is his assertion rather, that the two women have a confused sexual identity – the evidence for which is, that although "feminine and attractive in appearance", they have a phallic sexuality, and that this somehow gives rise to their need for triumphant retribution against those who are more naturally endowed in this way? Or does a phallic sexuality only become sadistic and exhibitionistic in the wrong hands, so to speak?

Without putting too fine a point on it, Dr Freeman has succeeded in spinning a great yarn about the origins of morbid jealousy in these women with precious little evidence that their jealousy was 'morbid'. What he described are two chaotic and unhappy marital relationships (marriage being an arrangement between two people, unless I am mistaken) where sexuality has become the bargaining chip and weapon, as it not uncommonly does for damaged and powerless people of both sexes.

Finally, it must be the women in the textbooks of psychiatry (or psychoanalysis) for whom the absence of vaginal orgasm is pathological, the onset of menstruation was not a shock (for those over 30), and the admiration of breasts bigger than one's own is a homosexual urge.

Freud was a man of undisputed genius, but he did not know (as distinct from presume) much about women – how could he? (See Peter Gay's new biography: *Freud–A Life for Our Time*.) I hope those who purport to heal psyches in the name of psychoanalysis are not following in his footsteps.

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Genetic origins of psychosis

SIR: Crow recently discussed the genetic origins of psychosis (*Journal*, June 1990, **156**, 788–797). We recently completed a retroactive case study of 574 patients suffering from schizophrenic psychosis. Our results showed that the incidence of schizophrenia increases with progressively increasing paternal age. The association between parental age and mental illness has often been reported in the past. Such reports have come from different parts of the world and have involved thousands of patients. Kinnell (1983) wrote: "There are few areas of schizophrenic research where one finds such impressive concordance of results from different studies as that of parental age". Our results showed that the age of the father is the operant factor. The association between paternal age and at least 11 genetic diseases or recent mutational origin has been documented. Evans (1988) wrote: "There is considerable and long-standing evidence that mutation frequencies for a variety of different human genes causing disease increase with increasing paternal age". Vogel & Rathenberg (1975) reported that the special sensitivity of the male germ cells for mutation increases with age.

It can be concluded that increased mutation rate in the germ cells of the aged father could play a role in the causation of schizophrenia in some cases. Schizophrenic individuals born to older fathers could constitute an aetiologically distinct subgroup of the schizophrenic population.

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Sir: Crow (*Journal*, June 1990, **156**, 788–797) discusses the hypothesis that schizophrenia results from a disorder in the gene which determines cerebral dominance. In conceptual form the theory is supported by analysis of the unifying feature of the first-rank symptoms. These refer to the reception of vocal input ascribed to an external source, passivity phenomena in which a controlling external agency is implicated in thought insertion or withdrawal, or the imposition of motor or sensory data. In thought broadcast, thoughts are felt to be available to another externally. The subjective experience of the non-dominant hemisphere in its interaction with a dominant vocal hemisphere explains the above if the unifying feature is the awareness of the involvement of an external agency. These symptoms are understandable when considered in systems control theory as the experience of a processing unit which achieves a relative independence and seeks to understand its controller (e.g. reception of information by the right hemisphere from the left would be seen as thought insertion and vice versa as thought withdrawal). The above system would conform to the requirements for a neuropsychology of schizophrenia described by Frith & Done (1988) if the monitor and plan-evolving centre with willed intention represent dominant hemispherical functioning, while synthesis of perception, stimulus intention and resultant action represent non-dominant functioning. Support for the above possibility is mentioned in Birchwood *et al* (1988), where an account is given of the absence of first-rank symptoms in split-brain patients, post-hemispherectomy or in those with agenesis of the corpus callosum.

While there is conceptual support for Dr Crow's hypothesis, in consideration of the phenomenology of schizophrenia, the ascription of the abnormality of cerebral dominance to a single genetic abnormality is questionable. The single strongest point of opposition is Badian's (1983) paper (quoted by Crow, 1986) concerning the seasonal incidence of left-handedness. The results showed an excess of left-handedness in males born between September and February, with a high statistical significance. This pattern of increased left-handedness correlates well with the observed excess of winter births in schizophrenia. However, for cerebral dominance to interact so closely with an environmental variable any genetic theory of dominance must invoke an intermediate stage in development which is subject to the effects of an environmental variable. I am presently researching the possibility that the ratio of light:dark in the first few months following birth affects the left hemisphere to a greater extent than the right, and that genetic sensitivity to its effect would explain the majority of the above findings. In the first few months following birth, with the four-fold increase in brain mass, there would be a particular neurodevelopmental susceptibility to the action of an environmental variable.

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Post-traumatic stress disorder

Sir: Kennedy (*Journal*, January 1990, **156**, 129) wrote questioning whether the data in my recent studies about Australian volunteer fire fighters is capable of generalisation to post-traumatic stress disorder (PTSD) as a whole. He suggests that this may not be the case because this group was trained, well motivated and had previous experience. He goes on to state that the event lacked the element of surprise and unfamiliarity experienced by other PTSD groups examined.

These are important issues to consider. However, the experience of these fire fighters in the Ash Wednesday disaster was far beyond their most extreme expectations. The intensity of the heat in these fires was very great, with five metres of fire-front generating as much energy as a large power station (Webster, 1986). Most of these volunteers were also members of the local community and many had their homes damaged or destroyed. As well, while they were fighting the blaze, most knew that their families were also facing considerable risk. This was particularly difficult for them because they were not in a position to protect their families. Furthermore, often the radio communications used by the fire networks were completely ineffective because the intensity of the heat ionised the atmosphere. This created an atmosphere of isolation and lack of direction. Moreover, much of the fire-fighting equipment also failed during the fires. Many of the vehicles used were powered by petrol motors, or had petrol pumps working the fire fighting equipment. The intensity of the heat was sufficient to vaporise the fuel in the lines. As a result they were often trapped in extraordinarily dangerous circumstances without equipment to protect themselves.

Having spoken to many of the fire fighters, this experience was both way beyond their wildest expectations of a major fire and had all the elements of extreme surprise and threat. Having interviewed many victims of the disaster (McFarlane, 1986) as well as studied the families of those victims (McFarlane *et al*, 1987), it appears that the experience of these fire fighters was often worse than that of the victims themselves because they were repeatedly exposed to extreme danger as well as having at times to remain on duty for periods of up to three days.

The experience of these fire fighters was thus one similar to all victims of extreme threat and danger. While they had some training, in the circumstances experienced, this offered them little or no protection. Therefore these data, I believe, are generally applicable to the aetiology and phenomonology of PTSD.

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Malaria presenting as atypical depression

Sir: Arun Prakash & Stein (*Journal*, April 1990, **156**, 594–595) mention hysterical stupor and atypical depression as sequelae to cerebral malaria. However, this seems disputable.

The World Health Organization (1986) specifically recommends to restrict diagnosis of cerebral malaria to patients with unarousable coma (showing non-localising or absent motor responses to noxious stimuli), in whom other causes of encephalopathy have been excluded. So, mild or transient cerebral dysfunction in a patient with malaria should not automatically be diagnosed as evidence of sequestration of parasitised erythrocytes in the cerebral vascular bed, which is the underlying pathophysiology of cerebral malaria (Osuntokun, 1985).

In addition, it seems improbable that at the time of admission the patient was actually suffering from malaria. In a non-immune individual not using antimalarial prophylactic medication, normal clinical as well as laboratory tests virtually rule out the diagnosis. Even in low-grade infections, splenomegaly, slight anemia and a raised erythrocyte sedimentation rate are obligatory (Manson-Bahr & Apted, 1982).

Plasmodium vivax malaria, which occurs in Thailand, may remain dormant for a long time, especially after insufficient chemoprophylaxis (Manson-Bahr & Apted, 1982). It can be reactivated among others by immunosuppression. A major depressive episode (Denman, 1986), malnutrition (Chandra, 1983) and, possibly, treatment with a tricyclic antidepressant (Denman, 1986) have been reported to impair immunocompetence. Therefore, it appears more likely that the malarial attack in this patient occurred in the course of and not before her depressive episode. Of course, this does not make treatment any less imperative.

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Anorexia nervosa in people of Asian extraction

Sir: Bhadrinath (*Journal*, April 1990, **156**, 565–568) presented three case-reports of anorexia nervosa in adolescents of Asian extraction which were very interesting. We are led to believe that the condition in non-white populations in the UK is very rare. Despite the small numbers of people from ethnic minority groups in the area of East Suffolk we have seen the condition in a teenage girl from a

Bangladeshi family. We also came across some problems with fasting at Ramadan. We agree with Dr Bhandrinath that there is a need for a better understanding of attitudes to body shape and food within different subcultures. We are frequently reminded that the second generation of children from ethnic minority families do not necessarily follow the views and values of their parents nor indeed that of the host culture in which they presently live. Although work has been done at looking at anorexia nervosa in black populations (Buchan & Gregory, 1984; Holden & Robinson, 1988), it would be interesting to look at the occurrence of anorexia nervosa and bulimia nervosa in British-born Asian patients.

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SIR: We read with interest the article by Badrinath (*Journal*, April 1990, **156**, 565–568), and wish to make some comments based on our experience with eating disorders in India. Although published literature is lacking, it is likely that these disorders are far less prevalent in India than in developed countries. However, more interesting is the finding that the presentation of eating disorders encountered in India also varies from the typical description of anorexia nervosa. Our patients are also young females showing decreased appetite, excessive weight loss and amenorrhoea. But a clear body image disturbance or fear of becoming fat is hardly ever seen. Hyperactivity, abnormal food handling and bulimic symptoms are also very uncommon. On the other hand, vomiting is a frequent symptom, often contributing significantly to the weight loss. Sometimes this presentation is accompanied by other functional or clear conversion symptoms (e.g. localised pain, aphonia, hysterical seizures). Most of these patients do not satisfy the necessary criteria of anorexia nervosa according to the classification systems like the DSM–III–R, although they share the central feature of refusal to maintain body weight over a minimum normal weight for age and height. Often these patients are placed in the residual category of eating disorders not otherwise specified.

The lesser prevalence as well as variation in presentation are both perhaps related to sociocultural factors. As Badrinath mentions, traditional Indian culture does not emphasise thinness as a must for feminine beauty. Neither is the concern with body image as much as that in the West. In contrast to anorexia nervosa, hysterical symptoms are commonly encountered in India (Saxena *et al*, 1986), while they have become rare in developed countries. It may be relevant here to refer to Bemporad *et al* (1988), who have discussed the similarities between anorexia nervosa and hysteria They postulate that both these seem to be expressions of age-specific conflicts intensified by constrictive cultural ideas. This may explain the inverse relationship between the prevalences of anorexia nervosa and hysteria in the developed and the developing countries. From this viewpoint, the occurrence of hysterical symptoms in Indian patients with anorexia nervosa may also not be entirely coincidental.

Indian culture is increasingly coming under the influence of Western norms and values. It will be interesting to observe whether anorexia nervosa becomes more prevalent as well as more typical in presentation in the coming years.

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Temporal lobe in schizophrenia

SIR: The role of abnormalities of temporal structure in schizophrenia (*Journal*, May 1990, **156**, 615–619) is supported by reports of temporal atrophy in schizophrenics evaluated by computerised tomography (Yates *et al*, 1990), right temporoparietal inefficiency in schizophrenics assessed during a continuous performance test by positron emission tomography (*Journal*, February 1990, **156**, 216–227), and reduction in the total volume of the gray matter in the left temporal lobe in the brains of monozygotic twins discordant for schizophrenia (Suddath *et al*, 1990).

Dysregulation of frontal function by abnormalities of temporal structure (*Journal*, May 1990, **156**, 615–619) may be due to inefficient cortical circuits and dopamine abnormalities in the right hemisphere (Friedman, 1990; Jankovic, 1990) whose activity level may be assessed in schizophrenia by regional cerebral blood-flow during cognitive tasks (Berman & Weinberger, 1990).

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A HUNDRED YEARS AGO

After-care of the Insane

The After-care Association of the Insane, of which the Rev. Henry Hawkins, Colney Hatch, is the Honorary Secretary, continues to carry on such excellent work, and is one so little likely to achieve ordinary popularity, that we feel bound to say a specially good word for it. During the past year fifty cases of female convalescents leaving asylums for the insane in a poor and friendless condition have been dealt with. In nearly every case assisted suitable employment was afterwards found. Convalescents have been boarded out in cottages with great success, under the superintendence of local friends who have rendered most valuable assistance in finding suitable employment for a large number of cases boarded out. The Earl of Meath, Dr Hack Tuke, and Dr T. Claye Shaw have been appointed trustees during the year. Cases have been assisted from eighteen different asylums. The Council feel that the warm co-operation and assistance of medical superintendents is vital to the success of their work, and it is sincerely hoped that many more will join the Society during the coming twelve months. Reprinted from the *British Medical Journal*, June 14th, 1890.

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Books Reconsidered

The Disease Concept of Alcoholism: E. M. Jellinek*

Concern over those individuals who drink alcohol to excess goes back many centuries, this concern being directed at the effect alcohol has on the individual and on society. Levine (1978) pointed out that the idea of alcoholism as a progressive disease with the key symptom of 'loss of control' did not simply start with the foundation of Alcoholics Anonymous or the publication of Jellinek's monograph. Such a concept is, in fact, at least 200 years old.

During the last century there was much debate on whether alcoholism was a *disease* or a form of *deviance* as well as on the issue of *control of drinking*. Levine (1978) emphasises that alcohol was regarded as an addictive substance long before opium was. The Temperance Movement, the main body concerned with alcohol problems during the 19th century, accepted the concept of addiction, but emphasised *alcohol* as being the addictive agent. However, following Prohibition, the disease model of alcoholism came more to the fore, stressing that the cause of addiction related to the individual host and not to alcohol itself.

Such an idea of alcohol as a disease had been advanced long before Prohibition. Rush (1814), for example, designated intoxication a disease of the will which resulted in increasingly frequent bouts of drinking. However, others in the 19th century such as Todd (1882; cited by Jellinek) argued that the individual did not lose control but rather chose to drink – "the man drinks simply because he likes to drink or because he likes to be drunk".

The contribution of Jellinek

Jellinek's publication was the principle landmark in the field of alcoholism this century. Jellinek, through a variety of methods including obtaining information from members of Alcoholics Anonymous and extensive reading of the literature, attempted to extract a disorder called alcoholism from a variety of drinking patterns that were known to occur. Together with the views of Alcoholics Anonymous, the academic support of the Yale School of Alcohol Studies and the development of the National Council on Alcoholism, this led to an almost universal acceptance of the disease concept in the USA, for many years.

In Britain the reaction was more ambivalent. Although the *British Journal of Addiction* (formerly the *British Journal of Inebriety*) was first published in 1892, the majority of physicians in the UK did not believe in the disease concept. The traditional British view was that alcoholism was nothing but drunkenness and bad behaviour which just did not occur among decent people. This ambivalence towards the disease concept in the UK is probably the prime reason why there has been relatively few specialised facilities provided to help people with alcohol problems over the last 30 years.

Jellinek defined alcoholism as "any use of alcoholic beverages that causes any damage to the individual or society or both". He particularly stressed two types of alcoholism (gamma and delta) which showed clinical features he considered indicative of a disease.

Features included in both types were acquired tissue tolerance to alcohol, adaptive cell metabolism, withdrawal symptoms on cessation of alcohol intake, craving for alcohol, and either the loss of control drinking pattern (gamma) or the inability to abstain (delta). In gamma alcoholism individuals would lose control immediately they started drinking, the ingestion of one drink leading to a chain reaction (i.e. drinking would occur in a mechanistic way), while in delta alcoholism the individual was unable to abstain even for a day or two from alcohol.

Jellinek was fully aware of cultural and economic factors that influence alcohol consumption, and was also concerned that an exclusive emphasis on the disease concept of alcoholism (gamma and delta alcoholism) would exclude many other problems related to alcohol. He also considered that certain other patterns of drinking were symptomatic, that is, carried out to relieve underlying symptoms, and that these were therefore not illnesses since they lacked the features of the gamma and delta patterns. Alcohol was an agent capable of inducing addiction, and in those with gamma or delta alcoholism it was likely that there would be a progression from psychological to physical addiction.

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To Jellinek, adaption of cell metabolism to alcohol was a feature which indicated physiopathological changes in body cells analogous to those changes believed to occur in drug addiction. While being aware of their theoretical (non-factual) nature, he extensively lists the different theories of such changes. He seemed to draw close parallels between patterns of behaviour in the gamma and delta subjects with those among individuals addicted to opiates. He also regarded opiate addiction as a disease and believed that just because an individual had such a disease which was a form of addiction, it was not outside his or her powers to do something about it. Such an individual was, therefore, responsible for taking the necessary steps to deal with it.

It is worth stressing the latter point as recent critics (e.g. Heather & Robertson, 1981) have discussed how labelling individuals as alcoholics may exclude them from responsibility and will also stigmatise them. These critics have also emphasised how such labelling may exclude others with drinking problems from attending treatment centres and deter individuals in the early stages of alcohol problems from attending for help. Furthermore, it may encourage the perpetuation of an irreversible drinking pattern and would thus be self-fulfilling.

Jellinek separated those factors responsible for drinking before the onset of the disease from the disease itself. He agreed that learnt behaviour may be an important factor in pre-disease drinking, but while understanding psychological formulations which saw drinking as a symptom of underlying disorders, did not consider these formulations themselves explained the process of alcoholism. Essentially, Jellinek's theory involves seeing an individual drinking for a variety of reasons (including social, psychological, and cultural), and in certain individuals this leads to a process (of as yet unknown physiological and pathological change) which explains the key features described above. Although genetic factors might be important, Jellinek suggested that any individual, however healthy or well organised, who drinks heavily enough for long enough may become addicted. The marker of physical dependence was the presence of withdrawal symptoms, and here Jellinek drew on the experimental work of Isbell *et al* (1955: cited in Jellinek), particularly those experiments in which former morphine addicts were given large amounts of alcohol and subsequent withdrawal symptoms were assessed on cessation of alcohol intake.

The World Health Organization (WHO; 1952) described addictive drugs as falling into three types: those where pharmacological properties of the drug were of primary importance (e.g. opiates); those habit-forming drugs in which the psychological reaction of the user was of primary importance; and those of an intermediate category where both factors were important. Alcohol was considered as a drug in the intermediate category: its pharmacological action was important but addiction could only occur in those individuals whose make-up led them to seek an escape in alcohol.

Adaptation of cell metabolism, one of the key features of alcoholism, was thought to be indicated by the presence of withdrawal symptoms. Another key feature, increased tissue tolerance, had been clearly demonstrated by tests which measured the threshold of blood concentration at which performance deteriorated. These showed that in heavy drinkers a higher blood alcohol level was required to produce this deterioration. Increased tolerance was regarded as a changed physiological response to alcohol, and it was suggested that this would also be combined with the inability of individuals to compensate for this, particularly on individual tasks. Physiological experiments on people had already demonstrated that tolerance could be induced, and it was known that alcohol was cross-tolerant with other drugs such as general anaesthetics – higher levels of anaesthetics were needed in alcoholics or heavy drinkers. It was also known that changes in tolerance could not be explained by the change in the rate of absorption of alcohol from the gut or increase in oxygenation or excretion.

Craving was regarded by Jellinek as being of two types: a psychological type which occurred in-between bouts of drinking and accounted for relapse after abstinence; and a physiological or potential physiological type which occurred during the time that people were drinking. These related to delta and gamma alcoholism respectively. It should be emphasised that loss of control to Jellinek, apart from meaning the loss of freedom following the first ingestion of alcohol in a new bout of drinking, was "characterised by minor withdrawal symptoms in the presence of alcohol in the blood stream and the failure to achieve the desired euphoria for more than a few minutes".

Implications of the disease concept

In discussing the attitudes of the WHO, Jellinek argued that whether alcoholism was regarded as an illness or not was not crucial; what was important was where it was placed. He also stated that there were many conditions that medical men legitimately claimed as their own which did not constitute diseases.

Although there were inherent difficulties in labelling a disorder as a disease, placing it within the sphere of health rather than that of morality was of major importance in helping alcohol abusers, particularly in the USA. Jellinek reviewed the attitude of the Federal Government and different states within the USA towards the concept of alcoholism as a disease. In one state, Rhode Island, alcoholism was *not* seen as a public health problem but rather was related to a legal act covering chastity, morality and decency! Jellinek stressed that the attitude of the public was crucial in helping to provide incentives for the rehabilitation of the alcoholic.

Criticisms of the disease concept

Before the onslaught of criticism by behavioural psychologists (e.g. Heather & Robertson, 1981), there were two other important developments. Firstly, there was Keller's (1972) comment that while a loss of control and the fact of one drink leading to continuous drinking could clearly happen in an individual, not all individuals lost control on every occasion and, in fact, many controlled their drinking for certain periods. Secondly, there was Edwards & Gross's (1976) description of the alcohol dependence syndrome, which was clearly influenced by Jellinek, but different in that the syndrome was regarded as a collection of signs and symptoms, and was graded, i.e. it was not an 'all or none phenomena'. Individuals could be at different levels of dependence and move in either direction. Edwards & Gross also considered that biochemical factors as well as psychological ones were important in describing the syndrome. However, as Heather & Robertson (1981) pointed out, particularly with the emphasis on the immediate loss of control of drinking after a period of abstinence, in many ways the alcohol dependence syndrome was a more socially acceptable form of the disease concept. They criticise both Keller (1972) and Edwards & Gross in their suggestion that "concepts which do not qualify as scientific propositions are those which are not falsifiable. The conception of impaired control is not falsifiable because it cannot specify the conditions under which controlled drinking will occur and the conditions under which it will not and for the purposes of scientific discourse, is strictly meaningless".

Jellinek's concepts have been most critically analysed by Heather & Robertson (1981) using a behavioural psychology perspective and a knowledge of statistics. They drew attention to important experimental evidence which had developed after Jellinek's book was published, including studies which attempted to determine the environmental correlates of designated behavioural responses in an individual. By changing environmental consequences, changes in responses were recorded. In relation to abusive drinking, the experiments involved alcoholics carrying out spontaneous tasks, the reward of which would be money or alcohol; in general, the harder the individual worked, the more reward he/she obtained. In this situation (which of course is different from that outside the laboratory) individuals did not drink rapidly and consistently to high levels of intoxication, and they frequently chose not to work harder to obtain more alcohol.

Heather & Robertson (1981) concluded therefore that in those individuals labelled alcoholics there was clearly the ability to control their drinking behaviour. In clinical experience, of course, many experienced doctors would agree that individuals diagnosed as alcohol addicts or suffering from the alcohol dependence syndrome do abstain for periods, and on some occasions only drink to moderate levels of intoxication. It is also clear that environmental factors, for example being in a group of other men who are drinking heavily, may govern the amount that the individual drinks. For Heather & Robertson (1981) alcoholics certainly did not show mechanical drinking to the extreme degrees of intoxication predicted by Jellinek's theories (i.e. "one drink leads to a drunk"), nor did they always show an inability to abstain.

A second group of experiments quoted by Heather & Robertson showed what happened to individuals when they were given priming doses of alcohol or when they thought they had received alcohol but had not. As with experiments on aggression and alcohol, these found that whether an individual craved alcohol or not was much more a function of if they believed they had received it rather than if they had actually done so. This would support a psychological rather than a physiological explanation of craving.

Heather & Robertson (1981) concluded that "the issue of impaired control in alcoholics may be profitably explored in psychological, sociopsychological or broad sociological terms and need not be confined to physiological or other internal events. The important implications of these alternative explanations are that none of them rest on a strict separation of alcoholics from non-alcoholics and that none of them would entail any suggestion of an irreversible process".

Biological aspects of alcoholism

Jellinek placed great emphasis on biological processes responsible for the behaviour of an alcoholic, although he had no understanding, in terms of scientific facts, as to what these were. Behavioural critics of the disease concept, however, do not really explain how these types of phenomena particularly changes in functional tolerance and the presence of withdrawal symptoms, can be explained in psychological terms.

Gross and his colleagues (1977) developed a method of quantifiably measuring the alcohol withdrawal syndrome, and also subjected the pattern of withdrawal symptoms to factor analysis. Their work suggested that, in an uninterrupted episode of drinking, critical cumulative blood alcohol concentrations rather than quantities or days of alcohol intake were related to the development of most features of the withdrawal syndrome. During a period of 4–6 days of alcohol intake in alcoholics, significant levels of the withdrawal syndrome tended to occur after drinking episodes in which the average daily peak concentration was greater than approximately 200 mg per 100 ml, and the average daily minimal concentration was greater than about 50 mg per 100 ml.

Gross (1977) stated "the withdrawal syndrome may be viewed as an indirect toxic effect of the sustained critical concentrations of alcohol, presumably a consequence of the underlying changes which produce physical dependence during the intake period. With a sufficient number of such episodes or prolongation of drinking over a sufficient period of time and an insufficient number and duration of non-drinking periods which could presumably permit recovery, there is evidence that some residual manifestations of withdrawal may persist for months and might even become irreversible".

Particularly important to Gross' view was thus that physical dependence once it has been induced can be more easily re-introduced. This 'carry over' of physical dependence suggests the persistence of physical changes, perhaps in the brain. Functional tolerance, defined by Gross (1977) "as the diminished effect of the same blood alcohol concentration on neurophysiological and behavioural responses was also shown, once it has been acquired, to be more readily re-acquired". Again, this 'carry over' of functional tolerance suggests long-lasting changes.

Trachtenberg & Blum (1987) have suggested that the physiological craving for alcohol may be the result of naturally occurring deficits of opiate-like substances as well as other neurochemicals. These deficits may occur genetically or as a result of long-term drinking, and further psychological factors such as stress can produce a chronic deficiency of naturally occurring enkephalins and endorphines. These theories would link genetic, psychological and biological factors in a theory of alcoholism. At present, these theories cannot be proven, although there seems to be growing evidence of biochemical changes in fundamental areas of cerebral functions (e.g. levels of neurotransmitters and opioid-like substances in animals and maybe humans) after regular alcohol use. This, together with the 'carry over' of functional tolerance and physical dependence would suggest that in those that consume alcohol over a prolonged period there are long-lasting biological changes. This links with Jellinek's view that some biological process was occurring in the body cells of alcoholics.

Conclusions

There is no doubt that Jellinek's book was of major importance in establishing abnormal drinking as a matter that lay within the sphere of health rather than deviance. It set the tone and continues to do so for the illness concept of alcoholism, particularly in the USA. The criticisms and research studies have suggested, however, that some of the more simplistic concepts (e.g. immediate loss of control) have had to be modified. Nevertheless, the recent increase in interest of a biological process underlying dependence and the studies of Gross and colleagues would suggest that a biological factor is involved, particularly in the 'carry over' of physical dependence and functional tolerance. Jellinek believed strongly in such an underlying biological process. Furthermore, as his critics have pointed out, Jellinek's views have been largely incorporated into the alcohol dependence syndrome, which is the predominate model used by workers in the alcohol field, particularly in the UK.

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Book Reviews

Editor: Sidney Crown

Computer Applications in Psychiatry and Psychology. Edited by David Baskin. New York: Brunner/Mazel. 1990. 127 pp. $23.95.

The use of computers within psychiatric research and practice is now struggling to leave its adolescence phase. This short, readable, multi-authored book may help speed this maturation. The book reports proceedings from a symposium held at the Albert Enstein College of Medicine in New York, and so its contributions are uneven. In the best chapter, Greist gives a very useful summary of computer uses for psychiatric diagnosis including computerised algorithms, the development of computerised consultation, and computer-administered and computer-assisted interviews. He goes on to discuss the fascinating, recent work from his own team using computers to carry out cognitive behavioural therapy.

Among the other contributions there are worthwhile emphases on the challenges raised by computer applications. User resistance, both by research and clinical staff, are frequently encountered during the first stages of computerisation. As several of the authors indicate, if the staff who collect and enter the data into the computer feel a sense of access to and ownership of the outputs of the system, then user resistance is found to be far less.

Two chapters, those by Kennedy and Gastfriend, describe methods of establishing computerised patient databases, and will be of wide relevance to clinical teams in Britain who are now setting up databases of 'at-risk' patients. Indeed, the extent of Ericson's example of the Integrated Clinical Information Abstract, used at the Institute of Living in Hartford, will bring more than a twinge of envy to psychiatrists struggling with similar problems in Britain.

The book is primarily intended for an American audience, and one would wish to see more emphasis upon applications for population-based case registers, the development of clinical audit and quality assurance systems, and use in a wider range of clinical assessment and mental state instruments. Even so, this is a useful reference book for libraries and will be frequently requested as we move towards computer literacy.

Graham Thornicroft, *MRC Training Fellow, Institute of Psychiatry, London*

Textbook of Psychiatry. Edited by P. J. V. Beumont and R. B. Hampshire. Melbourne: Blackwell Scientific Publications. 1989. 530 pp. £29.50.

This Australian textbook is comprehensive and reasonably up-to-date, covering a wide field of psychiatric theory and practice, and including extensive chapters on the contributions made to psychiatry by the biological sciences, as well as the developmental and social influences. However, there are several problems with this book. It is aimed primarily at the undergraduate, but is clearly too bulky, too long and too expensive for the average medical student's requirements. Many of the chapters are rather advanced and seem more suited to the post-graduate, with the book ideally being used as a reference text only. Indeed, the appeal to registrars is acknowledged by the publishers, except that it is also highly recommended to family doctors, other clinicians, psychologists, social workers, occupational therapists, psychiatric nurses and "others in the health services". (Who's left – the hospital cat?).

The opening chapters relate to the basic concepts of psychiatric classification, the history of the speciality, and clinical assessment. Most of this is presented satisfactorily, although some parts of the text tend to be rather formal and tedious, and that relating to psychopathology is confused and repetitive. Other chapters deal with all aspects of clinical practice and the basic sciences, with most of the authors handling their designated topics in an adequate, if uninspiring, fashion. The chapters relating to affective disorders, drug treatments, psychological assessment, personality disorders, crisis/adjustment reactions and substance abuse are particularly useful and well written. On the other hand, those dealing with schizophrenia, anxiety states, mental retardation, behaviour therapy and community psychiatry are too brief and limited in scope for a book of this size. The sections relating to liaison work and the interface between psychiatry and general medicine are inconsistent and overcomplicated in some parts, but superficial in others.

Although the text claims to be up-to-date and adheres rigorously to the DSM-III-R classification system, I was struck by two anomalies in the book. Firstly, the concept of an 'epileptic personality' is now largely discredited, but is propounded in the chapter dealing with organic disorders. Secondly, the child psychiatry section refers to 'childhood psychosis', despite the fact that this is an anachronistic term no longer included in DSM–III–R. An other minor criticism is that little use is made of clinical vignettes (except in the chapter related to anxiety disorders), even though these tend to be much appreciated by student readers. Also, the book is specifically related to Australian psychiatric practice, rendering parts of it of no immediate relevance to the British undergraduate.

However, the main drawback to books such as this, in which numerous authors contribute their own separate chapters, is that style and quality can vary tremendously within the text, despite the best efforts of the editors to present a reasonably consistent end-product. This book fails to avoid that trap, and although I was left with mixed impressions, my overall view of it was somewhat negative.

J. J. HART, *Senior Registrar in Psychiatry, Runwell Hospital, Essex*

Chronic Epilepsy, Its Progress and Management. Edited by MICHAEL R. TRIMBLE. Chichester: John Wiley & Sons. 1989. 260 pp. £38.50.

It is reasonably well established that most patients with seizures – perhaps 80% – respond favourably to anticonvulsant medication, achieving long fit-free periods and often complete suppression of attacks. For the unfortunate refractory minority obliged to attend outpatient clinics, often for the rest of their lives, while complex and often intoxicating combinations of medications are abortively juggled, the pressures are profound and the prospects often grim. A host of problems (personal, psychiatric, neurological and toxicological) prove taxing to patients, their families and their advisers.

This book, in which 20 experienced authorities tackle different facets of the problem, is most welcome. Chapters comprehensively cover topics such as pathophysiology, prognosis, severity, monitoring, the problems of childhood, post-traumatic epilepsy, cognitive hazards, and treatment strategies including the role of surgery. Each chapter is succinct, realistic and generously referenced.

In recent years there has been increased awareness and research of the social dimensions of the disorder and the chapter by Thompson & Oxley, based upon a recent survey of such social difficulties, provides specific proposals for prevention and remedy. I anticipate that readers of this Journal will find the chapter on epilepsy in developing countries particularly intriguing. Despite methodological difficulties, the accounts of denial, concealment, sensitivities, local attitudes and beliefs and the conflicts between traditional views of illness and Western medicine are critically reviewed. The sociocultural variants appear to be infinite. In the high Andes, although there may be few motor cars, horseborne transport still presents considerable problems; in many parts of the Third World all cooking is done over open fires such that epilepsy is often known as "the burn disease"; a man with three wives and 30 children still felt that post-epileptic impotence was the major problem arising from his epilepsy. This salutory reminder, of course, applies to every community including our own. that an individual's perception of his disease may be radically different to that of his neighbour.

All those concerned in the care of patients with severe epilepsy will gain from reading this splendid book on a most difficult subject.

GERALD STERN, *Senior Consultant, Neurologist, Middlesex & University College Hospitals, London*

Cognitive-Analytic Therapy: Active Participation in Change. By ANTHONY RYLE. Chichester: John Wiley & Sons. 1990. 267 pp. £25.00.

Any type of psychotherapy is very difficult to learn from a book, hence the proliferation of workshops, seminars and courses that surround every therapy – culminating in the years of personal therapy required to become a psychoanalyst. It is from his impatience with these and other rituals surrounding psychoanalysis that Ryle has developed his own hybrid therapy, which draws extensively from psychoanalytic and Kellyian theory, and to a lesser extent from cognitive and behavioural therapies. From this has come a new treatment, styled cognitive analytic therapy (CAT). This book is an attempt to combine a description of the origins and theories of CAT with a practical guide to treatment. However, just as CAT is an occasionally uncomfortable hybrid of several contrasting therapies, the book also suffers from attempting to combine both the theoretical and practical in one account.

The problems posed by synthesising such unwilling bed fellows appears repeatedly throughout the pages For example, CAT makes great use of well defined, sensible procedures such as self-monitoring, homework, collaboration between therapist and patient, hypothesis testing and the use of rating scales, all of which clearly owe their origins to cognitive therapy. At the same time, many of the target problems identified seem imprecise and ill defined, and the solutions proposed can occasionally be banal or impractical. Thus the pragmatism of cognitive therapy becomes diluted by the symbolism and metaphor of analysis, while the frequent use of 'as if' and 'either/or' explanations begin to pall after a while. Similarly, Ryle is clearly anxious to avoid the irritating tendency of many therapeutic schools to develop their own hermetic jargon to describe relatively simple psychological events, but such abbreviations as TTP, TP, PSM, SDR and RRP appear throughout, and the uninitiated reader soon bemoans the absence of a glossary. Nevertheless, there is much that is refreshing in this account, while certain features, such as 'prose reformulation' at the start of treatment and the 'goodbye letter' at the end are both novel and intriguing.

Perhaps Ryle's main achievement has been to develop a method of psychotherapy that can be applied to the real world of the National Health Service (NHS), and not just the luxuries of private practise, being time-limited, practical, and applicable to a wide range of emotional problems. This is its most crucial, and welcome, feature. Nevertheless, claims that 16 sessions of cognitive analytic therapy (or anything) can be effective in treating borderline personality disorder should be treated with some scepticism – patterns of thought and behaviour that can be so easily abandoned simply do not fit with one's views of personality disorder. One would require some formal outcome data before readily accepting such claims.

Overall, the book is at its best in two areas. Ryle provides serious and thoughtful criticism of much that is wrong with modern therapies, from the inactive but all-knowing analyst to the short cuts of some of the modern American gurus whom Ryle clearly finds distasteful. The other strong point is the detailed accounts of actual therapies in progress – the book is stronger on clinical description than data. However, the verbal fluency and articulacy of both patients and therapists makes one wonder just how valid is the claim that CAT reaches beyond the confines of other talking cures. It is the theoretical basis that makes the hardest reading, and I suspect that many will find it easier to appreciate Ryle's profound clinical insights in person than in print.

SIMON WESSELY, *Wellcome Training Fellow in Epidemiology, Institute of Psychiatry*

Suicide and Ethnicity in the United States. By THE COMMITTEE ON CULTURAL PSYCHIATRY, GROUP FOR THE ADVANCEMENT OF PSYCHIATRY. New York: Brunner/Mazel. 1989. 148 pp. $15.95.

The main source of interest in this book is the chapter on suicide among blacks in the US. After highlighting the paucity of hard data, and the sources of error in those which are collected, the authors lay out the basic statistical evidence on rate of suicide (and homicide) by age, sex and race. The importance of this chapter lies in its possible relevance to the UK, and it highlights the need for further studies in ethnic groups in this country.

The American black population has a clearly lower rate of suicide as compared with whites, but there are interesting variations which feed the discussion and hypotheses developed. Young black men have a much higher rate both than those aged over 65 and than young black women. Emphasis is placed on the very high homicide rate among urban black youths (29.7 per 100 000) compared with the white rate of 5.6 (US Bureau of the Census, 1987), and they question whether some of these violent deaths are not, in fact, suicides. The most frequent methods of suicide among blacks in the US in 1972 were jumping and using firearms.

Some authors focus on the concept of 'victim precipitated homicide' and attempt to explain the lower suicide rate of blacks in these terms, while addressing the issue of the high black-on-black homicide rate. There is also a suggestion that the overall suicide rate is higher in less densely populated areas, where there is a less coherent black community, but that the levels of homicide and violence are much higher in the overcrowded urban setting.

Suicide rates are increasing in both black and white sectors of the population in the US, and in the UK.

The ability to translate the American experience to Britain is limited. Clearly there are major differences, the most obvious being the overall population of blacks in the US. This might suggest that the suicide rate among the Afro-Carribean ethnic groups in this country may be higher, and, without being able to quote figures, the homicide level may be very much lower. The insights gained in the discussion are universal, however, with inherent racism of white-dominated cultural and health institutions, the marginalisation of the black community and the acculturative stress clearly laid out for discussion. This book is published by the Group for the Advancement of Psychiatry, one of whose aims is to collect and appraise data in the fields of psychiatry, mental health and human relations. Its members have "worked closely with anthropologists, biologists, sociologists, economists, educators, lawyers, nurses, psychologists, social workers and experts in mass communication, philosophy and semantics". This is no bad thing and it shows in the opening out of the debate on suicide to include the Black perspective.

DAVID ROY, *Consultant Psychiatrist, Goodmayes Hospital*

Hypnosis: The Fourth European Congress at Oxford. Edited by DAVID WAXMAN, DAVID PEDERSEN, IAN WILKIE and PETER MELLETT. London: Whurr Publishers. 1989. 282 pp. £45.00.

This volume contains about 40 contributions selected, from those presented at the conference, by a panel of experts including those in psychiatric practice. The book is roughly divided in half, the first section including the more theoretical aspects of the subject and the second relating to the use of hypnosis in specific disorders. This latter is of more general interest and although results are reported, they are often of a preliminary nature.

Of interest to psychiatry is the use of hypnotherapy as an adjunct to the cognitive approach in the treatment of depression, as well as its use in psychotherapy to influence the amount and content of dreams. Hypnotherapy has also been applied to anorexia nervosa and the irritable bowel syndrome. Its use for relief of pain during delivery, following severe burns and in malignant disease is reported as well as that in aiding dental procedures. There is a tendency for the method to be of value when patients are anxious and stressed. Attempts to determine other factors which will predict benefit include electrodermal responses and various electroencephalogram (EEG) parameters, but further research needs to be undertaken in this area.

There are three contributions on the teaching of hypnosis. Not unnaturally there is concern about the question of whether hypnosis can be used to produce anti-social behaviour. This is, however, addressed by only one contribution. The paper relates to sexual abuse occurring during treatment by lay therapists; the case histories of the clients are described in detail.

Sadly, although the editors selected the papers carefully, they have not provided a list of contributors, nor an index. This seems a considerable defect since a wide range of disorders are covered and many potential readers may find it difficult to locate particular subjects. Nevertheless, this volume gives an interesting insight into the current state of hypnotherapy.

D. F. Scott, *Section of Neurological Sciences,*
London Hospital

Altered States of Consciousness and Mental Health. Edited by Colleen A. Ward. London & New Delhi: Sage Publications. 1989. 316 pp. £29.50.

Most psychiatrists assume that any deviation from the fully conscious state of self-awareness with full control over one's thoughts is, at best, undesirable and, at worst, an indication of abnormality or illness. Thus, if so-called altered states of consciousness (ASCs), such as trance states, possession states and even hypnotic states, occur naturally, an illness may be diagnosed or the matter dismissed as 'cultural' – the result of some sort of primitive practice characteristic of inferior forms of civilisation or (in Europe) a hangover from medieval times. Most psychologists are unwilling to make clear distinctions between normal and abnormal states of consiousness since this smacks of the 'disease' or medical model, but still wish to be scientific and objective; consequently, clinical psychology takes a behaviourist approach to consciousness, studying its concomitants rather than the state itself. Anthropology has its own racism but has the advantage in a multi-cultural world of being free of the extreme forms of ethnocentricism characteristic of the other two disciplines.

This book is a collection of 14 papers by authors who are well known figures in the fields of anthropology, psychiatry and psychology, all with a common interest in cross-cultural work. The subject matter is considered in four parts: theoretical issues, questions of 'psychopathology', therapeutic aspects of ASC and finally alternative approaches to the study of ASC. The introductions to each part are excellent as is the main introduction. Difficult and controversial issues are explored, often illustrated by case studies.

This book may not break new ground but, in bringing together a varied selection of papers about phenomena around the topic of human consciousness, it is an important contribution to cross-cultural understanding of human thinking. Unfortunately, it fails to confront questions of racism in the study and interpretation of non-Western people and their cultures and so, by omission rather than commission, perpetuates racist conceptions of superior and inferior cultures. The book will undoubtedly be read by transcultural psychologists but I would recommend it strongly to *all* psychologists and psychiatrists interested in trying to widen the perspectives of their basically Eurocentric disciplines.

Suman Fernando, *Consultant Psychiatrist,*
Department of Psychiatry, Chase Farm Hospital,
Enfield, Middlesex

The Healing Spirit. Case Studies in Religion and Psychotherapy. By Paul R. Fleischman. London: SPCK. 1990. 288 pp. £8.95.

Some may find the title of this work off-putting. In fact it is a masterly exposition of existential psychotherapy, in the sense that everyone has some 'world view', some basic conscious or unconscious attitude towards life itself and themselves in particular, which lies at the root of their specific decisions in life. It is part of the very structure of their personality.

The author highlights 10 major 'religious' factors which lie in the background of all psychotherapy, some of which being vital in the healing process of every individual therapy. He does so with the minimum of case history, but with each one being vividly portrayed. At the same time he examines each of the 10 factors as found in the world's leading faiths.

Here he is examining religion not merely as an institutional affiliation, but as a structural aspect of personality. It differs from the philosophical self in that it seeks to *discover* meanings, not to create them; and it differs from the scientific self in that it seeks to discover *meanings*, not causal forces.

In this examination, he also illustrates the way in which each of these 10 factors may be, and indeed have been, interpreted to the detriment of both individuals and nations.

These 10 factors illustrate the existential needs which are basic to every individual. They are categorised as: witnessed significance; lawful order; affirming acceptance; calling; membership; release; world view; human love; sacrifice; and meaningful death.

This is certainly a fundamental manual for anyone who is interested in existential therapy and especially for any therapist who is faced with a patient who has 'religious' problems or is expressing their emotional problems in religious terms.

LOUIS MARTEAU, *The Dympna Centre, 60 Grove End, London*

Understanding Women in Distress. By PAMELA ASHURST and ZAIDA HALL; with contributions from GEORGE CHRISTIE, GILL GORELL-BARNES, JANE KNOWLES, MIKE PAWSON and DINORA PINES. London: Tavistock/Routledge. 1989. 237 pp. £25.00 (hb), £12.95 (pb).

This book attempts to relate women's history, roles and socioeconomic positions to common experiences of distress. A wide variety of case material is sensitively described. Case studies show common expectations and pressures which lead to distress, while illustrating major differences in experience.

Part 1 provides a context and includes chapters on women's role and identity. Part 2 explores: eating disorders; abuse; lesbianism; infertility; loss of a child; a child with an impairment; depression; widowhood; separation and reconstitution of families; single motherhood; and medical problems.

Feminist analysis of women's position has highlighted how theoretical perspectives – limited by the ways in which women are commonly seen and by the language in which women's experience is described – may perpetuate stereotypes. Unfortunately, despite the promise of the title, the authors have not used feminist critiques effectively in their commentary on women in distress.

They claim that language is the vehicle for psychotherapy, yet use pejorative, stereotyping titles such as "Barren womanhood", "Womanhood despoiled" and "The imperfect child". In a book on women's distress, use of the generic 'he' is an oppressive convention.

Roles generally portrayed in the book are very traditional: man's attachment to care and woman's to home are assumed; a competitive business lifestyle is seen to be in conflict with aspirations towards motherhood; and "the true feminine equivalent of male potency is fertility, the ability to conceive and bear a child. A woman who cannot do this feels a profound sense of failure".

Broad generalisations are made often without evidence. The chapter on "Lesbian womanhood" is one of the few (others are Knowles' "Single motherhood" and Gorell-Barnes' "Stepmotherhood") which tries to avoid overgeneralisation. It concentrates on the meaning of particular events for individual women, recognising lesbianism as a sexual preference, not a psychiatric disorder. Distress is explored as a response to social stigma. Yet the following chapter begins: "A woman's capacity to create, bear and nurture a child is the very esense of her womanhood".

Lack of recognition that language mirrors and reinforces subordination is paralleled by lack of theoretical rigour in many chapters.

In "Hungry womanhood" an unsubstantiated link is made between the family dynamics of anorexic young women and those of families where children are sexually abused. Research comparing adequate numbers of 'normal' families has not been done.

There is speculation in "Womanhood despoiled" about a mother colluding in sexual abuse "by going out to work in the evening". An exploration of economic circumstances could have been expected as well as an acknowledgement that theories of mothers' collusion are strongly contested.

The book tries to cover a wide range of topics, too complex to be dealt with briefly. It risks encouraging assessment based on sweeping generalisations and interpretations rather than exploration of the unique configuration of influences – socioeconomic, cultural, personal – important in the individual circumstances.

There are tantalising glimpses of therapies used. More details of these would have sharpened the focus on the case material which is the book's strength.

NORMA BALDWIN, *Lecturer, University of Warwick*

Anxiety and Depression: Distinctive and Overlapping Features. Edited by PHILIP KENDALL and DAVID WATSON. London: Academic Press. 1989. 519 pp. $49.95.

This is a multi-authored volume and almost all of the contributions are by psychologists. Its aim is to examine the relationship between anxiety and depression, one which every psychiatrist will have come to realise is complex and far from clearly understood. Throughout the book the emphasis is on psychological theories; an allegiance to cognitive therapy is declared early on.

The book is divided fairly conventionally into sections on theory, assessment and diagnosis, social factors and life events, and treatment. The content of these sections, however, are far from standard. For instance, the theoretical chapters are devoted to the American psychological literature, to the exclusion of almost everything else. Both the largest section (six chapters) on social factors and the section on treatment (which is entirely psychological) digress to a considerable extent from the stated aim of the book. The clinical chapters, however, are more balanced in their review of the literature, not withstanding certain notable omissions; they all address the separability of anxiety and depression in a fairly systematic way.

Overall, this book suffers from a familiar set of shortcomings: there is little constancy of theme developed since the book is multi-authored; the psychiatric literature is irritatingly neglected since the authors are all psychologists; and being American, the style is earnest, and dense, yet somehow insubstantial. While overall this is not a book that will have general appeal, some individual chapters are thorough, well referenced and provide useful reviews for psychiatrists with an interest in psychological theory and cognitive therapy.

PETER MCKENNA, *Consultant Psychiatrist, Fulbourn Hospital, Fulbourn, Cambridge*

Drinking and Casualties: Accidents, Poisonings and Violence in an International Perspective. Edited by NORMAN GIESBRECHT, RENE GONZALEZ, MARCUS GRANT, ESA OSTERBERG, ROBIN ROOM, IRVING ROOTMAN and LELAND TOWLE. London: Routledge. 1989. 444 pp. £30.00.

With contributions from 38 international authors and editors, this book is divided into five sections.

Section I reviews the relationship between alcohol and trauma and examines alcohol-related casualties in Latin America, Africa and Oceania. The latter is particularly interesting because of its geographical fragmentation into the geoethnic regions of Melanesia, Micronesia and Polynesia.

Section II examines conceptual issues and methods and argues for a systems model to understand the complex relationship between alcohol consumption and injuries. It examines the role of alcohol in accidents, poisoning and violence, and there is a useful chapter on the pharmacology and method of analysis of alcohol. The section ends with research into and registration of alcohol-related non-fatal casualties in Finland.

Section III reviews studies of national systems of alcohol-related casualty statistics in Australia, Finland, Canada, Poland and Mexico, and again stresses the importance of more uniform data and record keeping, with recommendations of how this could be achieved.

Section IV covers exemplary analysis, and a chapter deals with biological matters of alcohol intake among 4796 subjects in accidents in France (a reprint of a paper appearing in the *British Medical Journal*, 1986). Other chapters in this section examine accidents and injuries among treated alcoholics and their families; trends in alcohol and violent deaths in Norway; sex differences in alcohol-related casualties in cases of self-destructive behaviour; and opportunities and problems in conducting research on alcohol and casualties in developing countries, by giving five case studies (the Sudan, Nigeria, Costa Rica, Argentina and Mexico).

Section V concentrates on research and policy. It emphasises that research on alcohol-related casualties must be recognised as a political and social undertaking and hence a redefinition of research and the roles and responsibilities of researchers within a sociopolitical context is needed. A further chapter looks at the similarities and differences in the researchers' and policy makers' agendas and a postscript outlines future directions in research and policy.

An appendix deals with the alcohol dimensions in casualty reporting systems and another proposes a model establishing an alcohol-related casualty surveillance based on US data systems.

As a clinician I found that this book was informative and allowed further insight into the topics discussed. I would recommend it to psychiatric libraries as a source of reference. It is also reasonably priced.

NAS CHOUDRY, *Consultant in Adult Mental Illness, St Ann's Hospital, Poole, Dorset*

Problem Drinking (2nd edn). By NICK HEATHER and IAN ROBERTSON. Oxford: Oxford University Press. 1989. 359 pp. £5.95.

This is the second edition of a book for the intelligent layman first published in 1985. It is complementary to the monograph for the professional (*Controlled Drinking*, 1981) by the same author.

The fundamental tenet of the book is that problem drinking is best understood as a learned type of behaviour with biological phenomena playing a limited role. After critically reviewing the history of the disease concept and emphatically rejecting it, the authors examine problem drinking as a social-psychological paradigm, giving a useful summary of different types of learning behaviour and the practical consequences including treatment and professional strategy.

Changes to the first edition include an update on the controlled drinking controversy, reports on controlled studies on early intervention attempts to modify drinking behaviour, and political developments in relation to control policies within the UK.

The book remains a valuable and clearly written exposition of problem drinking from the social–psychological perspective, although at times one is suspicious of the authors' attempt to explain practically all abnormal drinking behaviour using this model alone. For example, nothing is said of the phenomena of tolerance and withdrawal symptoms emerging at the same level when drinking is commenced after abstinance.

Finally, in a field of endeavour where few specialised workers of any discipline show any professional interest in the subject, it seems a pity that the work and role of the specialised alcoholism treatment unit is so summarily dismissed. Under the new National Health Service contract, there will be quality assurance assessments and investigations of patients' levels of satisfaction. It will be interesting to see whether patients who attend such units are also so dismissive.

BRIAN D. HORE, *Consultant Psychiatrist, Alcoholism Treatment Unit, Withington Hospital, Manchester*

Benzodiazepines: Current Concepts – Biological, Clinical and Social Perspectives. Edited by I. HINDMARCH, G. BEAUMONT, S. BRANDON and B. E. LEONARD. Chichester: John Wiley & Sons. 1990. 332 pp. £32.50.

In retrospect, all organisations involved with benzodiazepines since 1970 should have employed the services of a public relations officer. The high profile of these compounds in the last ten years has ensured that little can be said or written about them without some eager beaver of a journalist reporting the information to a far wider audience.

It is possible to detect the influence of public relations in this book. Ostensibly, it is a hard-nosed look at benzodiazepines from the viewpoints of animal pharmacology and clinical practice. We move from the benzodiazepine receptor (Haefely) and its role in dependence (Nutt) through to pharmacodynamics and pharmacokinetics (Leonard *et al*). Differences between benzodiazepines are explored further by Hindmarch, mainly through human volunteer studies. We then read about the range of use of benzodiazepines from general practice through to forensic populations. There are also chapters on epidemiology of benzodiazepine use (Katschnig & Amering), and sociological (Bury & Gabe) and legal implications (Brahams)

The message that comes over time and time again throughout this book is that benzodiazepines are useful and effective drugs and far too much fuss is being made about their disadvantages. Even in the two chapters on abuse (Katz *et al*) and dependence (Rickels *et al*) the disadvantages of benzodiazepines seem minor when compared with their benefits. Even one of the widely accepted modifications to clinical practice following from recent research – that it is wise to discontinue long-term benzodiazepine treatment – is questioned, as "it is not at all clear that this is appropriate, much less necessary – at least until such time as it can be shown either that long-term use provides no benefit to these patients or that long-term use poses some particular risks that have not appeared to date "(p. 194). This advice is only countered by Birley in the preface, who argues for the opposite viewpoint that such patients should "try to take them (benzodiazepines) intermittently or stop taking them altogether".

I was left with the uneasy feeling that despite the independence of many of the contributors, the primary motivation behind this book was to 'soft sell' the benzodiazepines rather than to look at them dispassionately. Although the temptation is strong to compensate for the many tendentious statements that have been made about them in recent years (delightfully illustrated in Bury & Gabe's chapter), it should nonetheless be resisted. This book is rather like a solemn treatise on gunpowder, detailing its chemical properties and widespread usage for peaceful purposes, with just the occasional footnote here and there that it can sometimes be used to kill people when used by a few irresponsibles, who, tut-tut, could hardly be numbered among this book's readers.

PETER TYRER, *Senior Lecturer, St Charles Hospital, London*

Geriatric Psychiatry. Edited by EWALD W. BUSSE and DAN G. BLAZER. Washington, DC: American Psychiatric Press. 1989. 725 pp. £55.00.

It is ten years since the appearance of the *Handbook of Geriatric Psychiatry* edited by the same authors, and this new book adequately reflects what has changed, and what has not. It generally stands up well to sampling.

There are 26 contributors, all from the United States, and they include well known as well as less well known names. By and large they have done a good job. It is not fair to any textbook to judge it on its up-to-dateness in the neurobiology of the brain and particularly of dementia, for this subject moves so fast that any textbook must, by the time it appears, be well behind the front line; this one, however, does as well as any.

The wholly North American base of the book means that it is unlikely to become popular as a standard text here, although it does notice much, but not all, of the important literature from outside the United States. Astonishingly (for the authors are good friends of British psychogeriatricians), they seem to have missed out on the history of the development of psychogeriatrics as a specialty in this country. From the section on Britain in the last chapter, which reviews the future of geriatric psychiatry, it could not be guessed that this field had any definable existence yet in Britain, let alone that it was now a part of district services.

Readers of the earlier volume will see many similarities, and there is overlap of structure and of authorship. An important advance is that this book, by contrast with the earlier two-column small-print volume, has a larger full-page print which is more pleasant; and there is a delightful picture of two older ladies on the cover, which is intriguingly cryptic: are they sisters, twins, or mother and daughter? And what is it about the picture that compels the view that they can only be American?

TOM ARIE, *Professor of Health Care of the Elderly, Nottingham University, Nottingham*

Community Care: People Leaving Long-Stay Hospital. Edited by SHEILA SHARKEY and SARA BARNA. London: Routledge. 1989. 238 pp. £25.00.

This book is based on the multidisciplinary conference on community care organised by the psychology service and training section of Northumberland Health Authority in 1987. It is divided into three sections dealing with the political and historical background, planning therapies, and the evaluation of examples of good practice. It aims to highlight some of the issues involved in the running down of large institutions, and to offer help to those engaged in the process of resettling ex-patients in the community.

The first chapter by Short presents a summary of some of her work as chairman of the Parliamentary Select Committee for Social Services. Chapter two, a historical review by Shepherd, is a most stimulating paper and should be widely read. In chapter three, Blunden looks briefly at the problem of achieving effectiveness and quality of service and, in particular, draws lessons from commercial organisations and their customers. Heginbotham pursues this theme further in chapter four.

The second part provides examples of planning services for the elderly, the mentally ill and the mentally handicapped. The papers are detailed and could be of value to those acitvely engaged in planning changes in their own services but are less likely to appeal to the general reader. The third and longest part of the book is concerned with examples of good practice including evaluation. Once again it is likely to be of value chiefly to those actively engaged in service development.

The conference took place after the publication of the Griffiths Report on community care but before any government response. Although its material is now three years old it is still a useful source book and as such would be a worthwhile purchase for psychiatric libraries and for managerial bookshelves.

A. C. BROWN, *Consultant Senior Lecturer, Department of Mental Health*

St Andrew's Hospital, Northampton: The First One Hundred and Fifty Years (1838–1988). By ARTHUR FOSS and KERITH TRICK. Cambridge: Granta Editions 1989. 312 pp. £14.95.

At first glance this book would not be out of place on a coffee table – it is sumptuously produced and boasts 80 illustrations. But closer inspection reveals that with an historian and a St Andrew's psychiatrist as joint authors, a meticulous history of St Andrew's itself, and of English psychiatry in general, has been produced

We are supposed to learn from history, and can do so most easily when remote events echo today. Thus, any psychiatrist who imagines that problems with MIND are a recent phenomenon will be fascinated to read of the 'Alleged Lunatics' Friends Society'. Similarly, those who are involved in drawing up contracts between their hospitals and health authorities will enjoy reading about the attempts, ultimately in vain, to agree pricing with the county, after the passing of the 1845 Lunatics Act. And the comments of the Commissioners of Lunacy could, with little modification, be read as reports from today's hospital advisory service. History should also encourage us to look forward, but despite the tantalising subtitle, "The first one hundred and fifty years", the authors do not speculate on the next century and a half – perhaps wisely.

In the background of St Andrew's we find the management committee, and we may contrast the selfless contributions of the Spencer family over the whole 150 years with the petty squabbling of churchmen and local politicians over the last century, which had little to do with the welfare of the hospital. In the foreground we have the 10 medical superintendents – one criticism of the book is that a franker account of shortcomings and a fuller account of successes is given of the earlier superintendents. It would have been of great interest to have read a critical comparison of the medical contribution to management between St Andrew's and the National Health Service (NHS), since the former chose to be independent in 1948. Certainly the freedom St Andrew's has enjoyed to exploit its estate and the changing skills of its staff, together with its record of identifying service gaps at national level and filling such niches, leaves those like me who are constrained by NHS bureaucracy very envious.

With the current upsurge of interest in the history of psychiatry and the College's involvement in the publication of a recent volume and the launch of a new journal, I warmly recommend that this fine book should grace every psychiatric library.

DAVID TAIT, *Physician Superintendent, Murray Royal Hospital, Perth*

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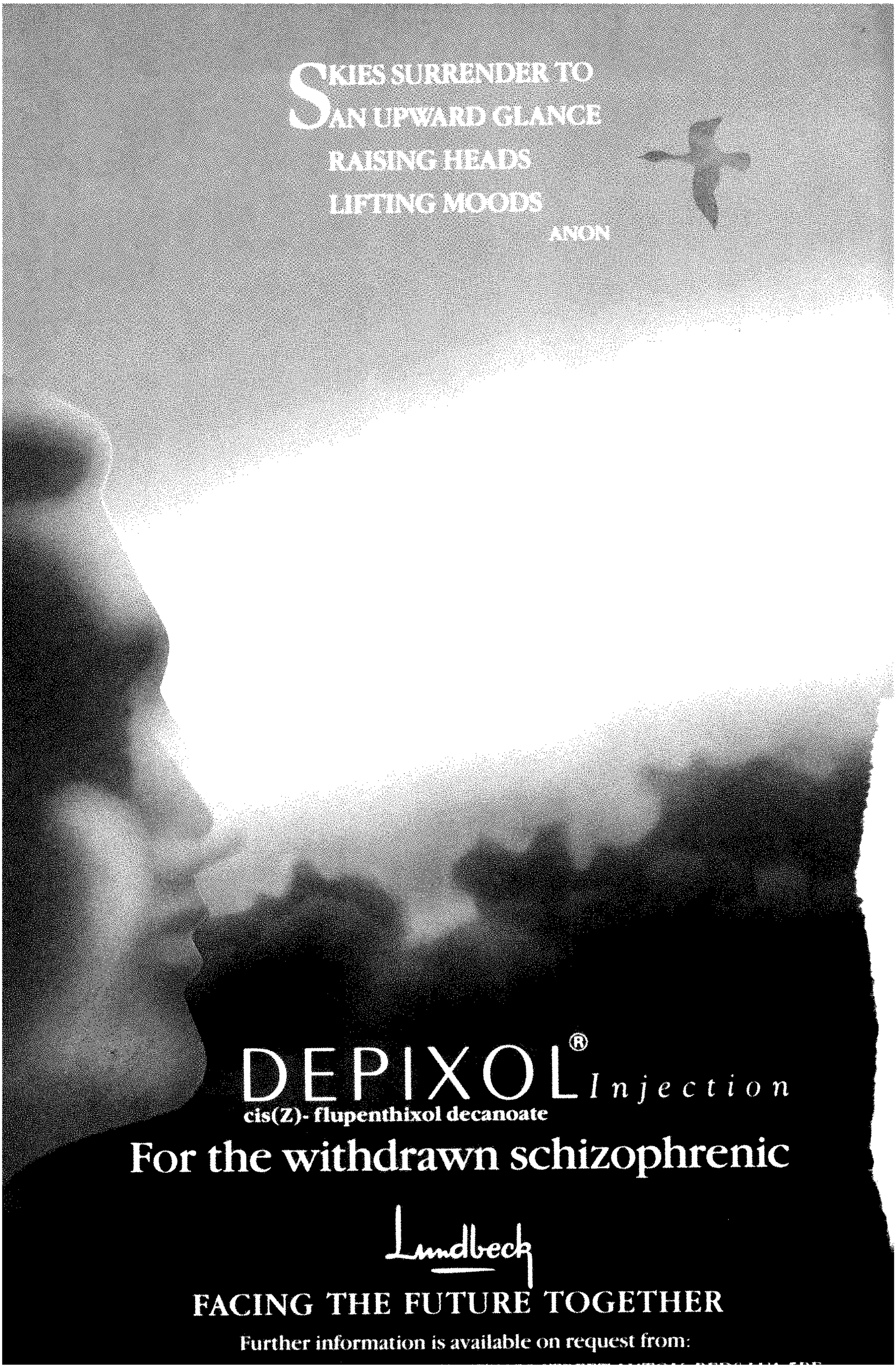

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Review Article

The Effect of Neuroleptics on Cognitive and Psychomotor Function

DAVID J. KING

There has been great variability and inconsistency in the reported effects of neuroleptic drugs on cognitive and psychomotor function in both patients and normal controls Experimental design rather than any particular cognitive or psychomotor test appears to have determined the sensitivity of detection of neuroleptic drug effects. In general, sedative phenothiazines have been found to depress psychomotor function and sustained attention, but higher cognitive functions are relatively unaffected. In the majority of studies of schizophrenic patients, both cognitive function and attention improve with neuroleptic treatment, in parallel with clinical recovery. Negative symptoms are not increased and usually show slight improvement with neuroleptic treatment. Controls are more sensitive than schizophrenic patients to neuroleptic drug-induced impairments. Tolerance has been seen in patients but has not been demonstrated in normal volunteers. The way in which neuroleptics produce their beneficial effects in patients remains unknown. Three main hypotheses to replace early arousal theories are proposed: normalisation of attention, facilitated indirectly by suppression of 'released' limbic dopamine hyperactivity; normalisation of asymmetrical temporohippocampal function; or direct improvement of attentional processing. Studies of the effects of new antipsychotic drugs with selective actions and the development of more reliable and selective tests of psychomotor and cognitive functions are required.

Studies of the effects of neuroleptics are important both to assess the nature and extent of their adverse effects and to elucidate their mode of action in psychosis. Inferences based on their biochemical actions led to the well known dopamine hypothesis of schizophrenia (Randrup & Munkvad, 1972; Klawans *et al*, 1972; Matthysse, 1973). Direct evidence for an abnormality at this level, however, is not yet convincing. On the other hand, evidence for psychophysiological abnormalities such as disrupted eye tracking (Diefendorf & Dodge, 1908) and prolonged reaction time (Rodnick & Shakow, 1940) in schizophrenia have long been established. Primary disorders of attention or arousal were first posited by McGhie & Chapman (1961) and Venables & Wing (1962), respectively. We therefore need to bridge a number of gaps between our understanding of biochemical pharmacology and the physiological mechanisms underlying abnormal cognitive processes and clinical symptoms. An understanding of the effects of neuroleptics on psychomotor and cognitive function, in both schizophrenic and normal subjects, should, therefore, lead to useful models for the pathophysiology of this condition. Both the speculations and experimental findings in the literature, however, have been inconsistent. On the one hand, Tecce & Cole (1972) maintained that neuroleptics improve cognitive function either by decreasing arousal or increasing attention, while on the other it has been proposed that neuroleptics have been responsible for the cognitive impairment found in schizophrenia (Marsden, 1976; Lidsky *et al*, 1981; Reus, 1983; Goldberg, 1985*a*).

In order to examine the evidence for these points of view, a review of the literature of reported studies in both patients and normal healthy volunteers was undertaken. A computer search was carried out on the Medline database, from 1966 to the present time, using 'major tranquillising agents', 'psychomotor performance' and 'cognition disorders' as key words. Earlier studies were traced from references in the later papers. Since the intention was to form an overview of the findings since these drugs were first used, no attempt was made to select studies by methodology, which has changed substantially over the past 35 years. The literature was classified first according to the subjects studied (patients or normal volunteers) and second according to the main drug investigated.

Studies in schizophrenic patients

Studies of neuroleptic drug effects can be divided into three groups: (a) those reporting no change or variable effects (ten studies), (b) those reporting a deficit in performance and cognitive function (six studies), and (c) those reporting improvement (14 studies).

(a) The earliest study appears to be that of Lehmann & Hanrahan (1954), who found variable effects on tests of reaction time (RT), hand steadiness, tapping speed, digit span and digit symbols in eight patients given "a dose of chlorpromazine sufficiently large to promote definite somnolence", in contrast to the uniform impairments of performance in these tests after secobarbital. With chronic treatment they observed that patients "are capable of sustained attention, reflection, and concentration", and, since these higher cortical functions were so little affected, they located the action of chlorpromazine in the activating reticular formation or hypothalamus. Five studies using simple RT measures (Heilizer, 1959; Pearl, 1962; Pugh, 1968; Held *et al*, 1970; Sakalis *et al*, 1972) and one using the Halstead–Reitan neuropsychological test battery (Howard *et al*, 1975) also reported no change with phenothiazines. A decade later Berman *et al* (1986) reported similar results. They were able to measure regional cerebral blood flow (rCBF) while patients carried out the Wisconsin Card-Sorting Test (WCST), a number-matching test (NM) and the continuous performance test (CPT). The WCST was believed to be a relatively specific test of frontal activity. They found reduced rCBF in the dorsolateral pre-frontal cortex (DLPFC) of both medicated and non-medicated patients, compared with controls, while they attempted the WCST but not while performing the other tests. They concluded that neuroleptics had little effect on DLPFC function or metabolism, and this was attributed to the paucity of autoreceptors in the mesocortical dopamine system. A further study also failed to detect any significant effect of haloperidol, flupenthixol or clozapine on sensorimotor or cognitive performance on a test battery which included the Stroop test (Classen & Launx, 1988). Although Tomer & Flor-Henry (1989) found no overall effect on cognitive performance in patients given a variety of antipsychotic drugs, they found that attention asymmetry was reversed, such that inattention on the right was improved at the expense of inattention on the left.

(b) Some early studies reported negative effects of acute or high doses of neuroleptics but with chronic administration tolerance developed and little or no effect was demonstrable (Kornetsky *et al*, 1959; Judson & MacCasland, 1960; Latz & Kornetsky, 1965). In fact, when Kornetsky (1972) reviewed his series of studies with the CPT he found that, while single doses of phenothiazines impaired performance, after chronic administration performance was improved. Erickson *et al* (1984), however, have reported impairment on the CPT in adolescent schizophrenics 35 days after treatment with low-potency sedative neuroleptics (thioridazine and thiothixine). Porteus (1957) found a decline in scores in his maze test in 15 of 22 patients treated with chlorpromazine (300 mg daily for four months) and, since these scores were similar to those after frontal lobotomy, he thought chlorpromazine acted on thalamocortical connections or the reticular formation to reduce anxiety at the expense of impaired vigilance. Cleghorn *et al* (1990) also reported impairments in Porteus maze performance as well as recall of complex designs (Rey figure) in 37 chronically medicated patients compared with 27 drug-free patients. The lower symptom ratings in the drug-free group in this study were associated with improvements in comprehension and short-term verbal recall but not with other neuropsychological measures.

(c) The majority of studies, however, report improvement in attention or cognitive function with neuroleptic treatment (Shatin *et al*, 1956; Brooks & Weaver, 1961; Court & Cameron, 1963; Remr, 1970, 1971; Orzack & Kornetsky, 1971; Serafetinides & Clark, 1973; Spohn *et al*, 1977; Gruzelier & Hammond, 1978; Oltmanns *et al*, 1978; Wahba *et al*, 1981; Walker, 1981, 1983; Braff & Saccuzzo, 1982; Marder *et al*, 1984; Saletu *et al*, 1986), although in two of these the changes were not statistically significant (Brooks & Weaver, 1961; Walker, 1983). Generally speaking, improvement paralleled clinical recovery (Spohn *et al*, 1977; Wahba *et al*, 1981) but not all these studies included assessments of the severity of clinical psychopathology. The study by Braff & Saccuzzo (1982) used a backward visual-masking task in which patients had to detect letters which were immediately followed by a masking stimulus of greater luminance and duration. Twenty medicated patients performed significantly better than 16 drug-free patients in this test when the interstimulus interval between target and mask was 120 or 300 ms. It was therefore concluded that neuroleptics accelerated information processing in schizophrenic patients. A similar finding had been reported in normal volunteers using a visual short-term memory task by Stone *et al* (1969) (see below). Saletu *et al* (1986) studied the effects of acute and chronic (six weeks') treatment with haloperidol or fluperlapine (an atypical neuroleptic resembling clozapine) on 12 cognitive and psychomotor tests, as well as smooth-pursuit eye movements and a number of electroencephalographic (EEG) measures in 20 schizophrenic patients. Both acute (5 mg) and chronic (20 mg per day) haloperidol treatment impaired fine motor performance, but chronic treatment improved cognitive function and subjective well-being. While single doses of fluperlapine (100 mg) impaired performance on a number of tests, after chronic treatment (500 mg per day) there were improvements in nine of these, covering cognition and psychomotor performance, as well as in subjective well-being.

In a critical review Medalia *et al* (1988) classified the reported effects of neuroleptics according to their effects on nine different neuropsychological functions. They concluded that the various cognitive functions were differentially affected. Thus impairments in memory and fine motor co-ordination were most consistently found and were probably related to anticholinergic effects and dopamine-receptor antagonism respectively. (However, while Cleghorn *et al* (1990) have also subsequently reported poor motor performance, they found improved verbal recall.) There was also a suggestion that planning, as measured by various maze tests, was also impaired, implying an adverse effect on frontal lobe function. However, tests of language, intelligence, and attention (including various RT measures and the CPT) as well as the Halstead–Reitan test battery gave equivocal results. On balance, no change was found on cognition or tests of visual–motor co-ordination, including trail-making and digit-symbol substitution tests (DSST). They found no consistent evidence for improvement in any neuropsychological test (the studies by Braff & Saccuzzo (1982) and Saletu *et al* (1986) are notable exceptions, but these were not included in the review by Medalia *et al* (1988)) and argued against the traditional notion of hyperarousal in schizophrenia, which was thought to be reduced by neuroleptics, leading to improved attention.

Neuroleptics and negative symptoms

Crow (1980) drew attention to the relative resistance of negative symptoms (poverty of thought, affective blunting, loss of drive and volition, and social withdrawal) to neuroleptic treatment and proposed a 'type II syndrome' to indicate that they may have a different aetiology from positive symptoms (delusions, hallucinations and thought disorder). He further suggested that negative symptoms were a later development of the disease, associated with cognitive impairment and increased cerebral ventricular size. Furthermore, as noted in the Introduction, some authors have implicated neuroleptics in the cause of cognitive impairment (Marsden, 1976; Lidsky *et al*, 1981; Reus, 1983; Goldberg, 1985*a*).

Nevertheless, as Freeman (1982) pointed out, negative symptoms should properly be regarded as primary rather than secondary, as in Bleuler's original concept, as they frequently precede positive symptoms. This idea derives from Jackson's (1889) model of the central nervous system (in which positive symptoms are essentially release phenomena) and was adapted in psychological terms by Ey (1962). It has most recently been expressed in pathophysiological terms by Weinberger (1987), who has proposed that lesions of the DLPFC produce negative symptoms, and positive symptoms are a result of failure to suppress stress-induced subcortical dopamine hyperactivity. According to these theories neuroleptics act at a subcortical (mesolimbic) level to reduce positive symptoms, but could not affect the 'primary' lesion causing negative symptoms.

Several studies, however, have found that negative symptoms do, in fact, respond to some extent to neuroleptic treatment (Goldberg, 1985*b*; Meltzer *et al*, 1986; Breier *et al*, 1987; van Kammen & Boronow, 1988; Kane & Mayerhoff, 1989). It may be of course, that dose levels are important in determining the net effect of neuroleptics on negative symptoms (Baldessarini *et al*, 1988). Indeed, Asarnow *et al* (1988) have shown that while schizophrenic patients with poor information-processing abilities do better over two years on a standard dose (25 mg every two weeks) of fluphenazine, the reverse is true of patients with good information-processing ability. The measure of information-processing ('span-of-apprehension' task), however, seemed to be a trait marker and was neither improved nor impaired by drug treatment.

A competing hypothesis is that positive and negative symptoms are related to deficits in function of left and right hemisphere, respectively (Gruzelier *et al*, 1988). There is some support for neuroleptic-induced reversal of such deficits (Tomer & Flor-Henry, 1989).

This issue cannot be resolved at present since decisive pathological evidence for the localisation of a fundamental lesion is not forthcoming (Andreasen, 1986; Grossman *et al*, 1989). The Jackson (1889) and Ey (1962) models do not require structural localisations, however, since they were thinking in terms of a functional hierarchy. Thus neuroleptics may, indeed, primarily act to suppress essentially normal, 'release' phenomena. There is, however, no direct evidence to suggest that negative symptoms are caused or exacerbated by neuroleptic drugs.

Cognitive function and tardive dyskinesia

In tardive dyskinesia (TD) there have been consistent reports of decreased cognitive function, dating from Edwards's (1970) survey of 184 long-stay female patients. He found a higher incidence of brain damage (signs of an organic brain syndrome or poor performance in a word-learning test) among 34 elderly patients with persistent oral dyskinesia compared with controls matched for age, sex and neuroleptic intake. Eight subsequent studies (Famuyiwa *et al*, 1979; Struve & Willner, 1983; Waddington *et al*, 1985; Wegner *et al*, 1985; McGuire & Thomas, 1986; Wade *et al*, 1987; Sorokin *et al*, 1988; Meise *et al*, 1988) reported similar findings. While two of these attributed the association to putative neurotoxic effects of neuroleptics (Famuyiwa *et al*, 1979; Wade *et al*, 1987), the majority agree with Waddington (1987) that the cognitive deficit reflects a pre-existing vulnerability to the development of TD. Waddington supports this contention by citing a number of studies that have shown that TD and non-TD patients are distinguishable by cognitive impairment, negative symptoms and cerebral ventricular enlargement but not by histories of neuroleptic drug exposure. Indeed, Struve & Willner (1983) have shown that cognitive impairment precedes the development of TD. Three studies have reported no cognitive changes with TD: in two of these, patients with cognitive disorder, dementia or cerebral atrophy were specifically excluded (Collerton *et al*, 1985; Myslobodsky *et al*, 1985) and in the other, patients over 60 years or with evidence of organic brain disorder were excluded (Marsalek *et al*, 1988). The appearance of TD is thus determined not only by neuroleptic exposure but also by the presence of a number of risk factors such as advancing age, female sex (Kane & Smith, 1982) and the presence of schizophrenic negative symptoms including cognitive impairment (Waddington & Youssef, 1986; Waddington, 1987).

Other clinical studies

There have been no consistent findings in studies with other psychiatric patients. Gottschalk & Cohn (1979) compared the effects of haloperidol with diazepam (both given intramuscularly in hourly doses of 5 mg for up to four hours) on the cognitive performance of alcoholics during detoxification. In spite of clinical improvement during this period, cognition failed to improve with either drug and the effects of haloperidol were variable, with improvements observed in some patients and deterioration in others. In a comparison of chlorpromazine, imipramine, phenelzine and diazepam with placebo in 180 depressed patients, Raskin *et al* (1983) found abstracting ability improved with chlorpromazine after three weeks, but this could not be distinguished from placebo and seems to have been due to the accompanying improvement in depression, although data on this were not provided. Conversely, in aggressive children, mild impairments on the Porteus maze test and simple RT tests were found after four weeks' treatment with haloperidol, but not lithium, in comparison with placebo (Platt *et al*, 1984). In a study of the effect of haloperidol on cognitive shifting ability, Berger *et al* (1989) found that three days after 60 mg of intramuscular haloperidol decanoate, 17 spasmodic torticollis patients had impaired performances on two card-sorting and two word-fluency tests compared with their drug-free scores. Controls matched for age and intelligence showed no change over a similar three-week interval. These data indicate that neuroleptics impair cognitive shifting but are the converse of those reported by Clark *et al* (1989) using a covert attentional orientation task (see under 'Droperidol' below).

Normal volunteers studies

In order to avoid the confounding effects of illness it is necessary to establish what cognitive and psychomotor effects these drugs have in norma healthy volunteers, notwithstanding the well known differences in tolerance to these drugs between psychiatric patients and normals, and the likely differences in the effects of single and chronic dosing.

(a) Phenothiazines

(i) Early studies

Chlorpromazine-induced impairments on tests o psychomotor and cognitive function and of sustained attention were demonstrated in a series of studie from the National Institutes of Mental Health between 1957 and 1965. Kornetsky *et al* (1957) firs studied the effects of two single doses of meperidine secobarbital, chlorpromazine and LSD in ten normal volunteers and reported that chlorpromazine and secobarbital caused similar impairment at a 200 mg dose on motor (pursuit-rotor) and psychomotor task (DSST and symbol-copying test), but the effects o neither drug were detectable on simple intellectual and perceptual tasks (addition and visual discrimination). There was a poor correlation between objective and subjective effects and they suspected that there were wide individual variations in response owing to th 'specific reactivity' of the subject. In order to reduc these they repeated their studies using only two drug (chlorpromazine and secobarbital) in 12 subjects They then found that, while chlorpromazine caused greater effects than similar doses of secobarbital on tests of motor co-ordination (pursuit-rotor, hand steadiness and tapping speed), secobarbital had greater effects than chlorpromazine on cognition

(DSST) (Kornetsky & Humphries, 1958). On the other hand, chlorpromazine had greater effects than equivalent doses of secobarbital on a CPT requiring sustained attention (Mirsky *et al*, 1959). Kornetsky & Orzack (1964) demonstrated that the chlorpromazine effect on the CPT was abolished if the task was 'subject paced' and sustained attention no longer necessary, and, similarly, that the secobarbital effect on a DSST was not detected if the cognitive component was removed, as in the symbol-copying test. These studies were summarised in a review by Mirsky & Kornetsky (1964), in which they concluded that whereas secobarbital caused more cognitive effects than chlorpromazine, chlorpromazine had greater effects than secobarbital on sustained attention. They therefore proposed that chlorpromazine acted preferentially on that portion of the ascending reticular activating system responsible for *tonic* (sustained) cortical arousal as opposed to *phasic* (brief) cortical arousal. Thus cortical function would be relatively spared and phasic arousal could be increased, via descending corticoreticular pathways, for the performance of brief cognitive tasks (DSST) but not for tasks which required more sustained (tonic) attention (CPT).

These effects were then compared with those in schizophrenic patients, and interesting similarities and differences found. While secobarbital impaired the performance of schizophrenics and normal subjects to the same extent, chlorpromazine (100 mg and 200 mg) caused more impairment in normals than in patients on most psychomotor tests (Kornetsky *et al*, 1959). Indeed, this differential effect was such that some differences in performance between schizophrenics and normals were reversed by chlorpromazine. Thus, in two tests (pursuit-rotor and tachistoscopic number recognition), although the performance of both normals and patients was impaired by chlorpromazine, normals were superior to schizophrenics after placebo but worse than schizophrenics after 200 mg chlorpromazine. In other tests (DSST and tapping speed) no such differential effect of chlorpromazine was found between patients and normal controls. Tolerance to chronic (11-day) administration of chlorpromazine, but not of secobarbital, was demonstrated in patients.

In another study Latz & Kornetsky (1965) found that the peak effect of secobarbital occurred 30–60 minutes after ingestion, but that of chlorpromazine occurred two to four hours after drug intake. Although schizophrenic patients performed significantly more poorly than normals on the CPT and DSST, at the time of peak effect the differential effects of the drugs on these tests was similar to that in normals, with chlorpromazine (100 mg and 200 mg) having a greater impairment on sustained attention (CPT) and secobarbital (200 mg) on cognition (DSST). Thus it appeared unlikely that clinical improvement could be brought about by a direct drug effect on attention. The improvements in attention reported in schizophrenics treated with phenothiazines, in the studies reviewed above, would seem, therefore, to be due to the combined effects of tolerance (Kornetsky *et al*, 1959) and clinical recovery.

(ii) Personality factors

The problem of wide variation in response because of personality factors was first raised by Kornetsky *et al* (1957) and was addressed in two studies. Kornetsky & Humphries (1957) measured personality factors in conjunction with the psychomotor tests in their first study (Kornetsky *et al*, 1957) and found that there were some correlations between scores on certain MMPI scales and both objective and subjective measures of impaired performance. Their data suggested that there were 'reactors' and 'non-reactors' to drugs and that reactors were more likely to be depressed and/or to have more unreasonable fears and to over-react to environmental stimuli. However, if the drug effect was marked enough, personality factors ceased to be important. Heninger *et al* (1965) also used MMPI scales and divided their normal subjects into type A and type B approximately corresponding to extraverts and introverts respectively. They also found both subjective and objective differences between drug effects in these two groups. While tranquillisation and improved rapport occurred in introverts after chlorpromazine or trifluoperazine, there was decreased rapport and increased apprehension in extraverts. Chlorpromazine (100 mg and 200 mg) caused cognitive impairment which was greater in extraverts than introverts, but while trifluoperazine (8 mg and 16 mg) caused psychomotor slowing in both types, nonsense syllable learning was improved in introverts. The interaction of personality, task difficulty and drug effects was further investigated by Janke & Debus (1972) with pimozide and haloperidol (see below). Evidently this is an area that requires clarification, particularly for studies of the effects of relatively low, single doses in normals.

(iii) Further studies with chlorpromazine

Other groups have studied chlorpromazine effects using different test strategies. Loeb *et al* (1965), using a prolonged (one-hour) auditory vigilance task, showed that amphetamine prevented a decline in performance over time, but chlorpromazine (50 mg) and placebo were indistinguishable on three of the four measures of vigilance. Besser & Duncan (1967) used thresholds on the auditory flutter fusion test (AFFT) and the visual critical flicker fusion test (CFFT) to plot the time course of sedative drug effects in 29 volunteers. They found that the effect of chlorpromazine (50 mg) was maximal at four hours and persisted for eight hours, which was consistent with the CPT and DSST data from Latz & Kornetsky (1965) (see above). Tecce *et al* (1975) were also able to demonstrate a similar time course of chlorpromazine (50 mg) in 28 subjects using an EEG measure (the contingent negative variation), which corresponded to the drug effects on simple RT tests. These studies suggest that the 100 mg and 200 mg doses employed by Kornetsky & Humphries (1958) and DiMascio *et al* (1963*a*, *b*) were unnecessarily high to demonstrate the effects of chlorpromazine, if enough subjects are used.

Hartley (1983) has reviewed a series of studies carried out by an Australasian group using 12–24 volunteers per study. They first showed that while both chlorpromazine (25 mg and 75 mg) and noise separately impaired performance on a one-hour visual vigilance task, there was no impairment when chlorpromazine and noise were applied together (Hartley *et al*, 1977). At that time they concluded that both noise and chlorpromazine independently increased the arousal of the brain-stem reticular formation, but interacted when given together. It could also be concluded, however, that noise caused supra-optimal and chlorpromazine sub-optimal arousal and that these actions cancelled each other out when noise and chlorpromazine were applied simultaneously. Hartley & Couper-Smartt (1978) demonstrated impairment with chlorpromazine (50 mg) on a visual integration test, no drug effects on a Stroop test, and an apparent stimulant effect of low-dose chlorpromazine (25 mg) in the Baddeley reasoning test. A further ingenious study using a prolonged (30-minute) choice reaction-time (CRT) test sought to distinguish between the effects of chlorpromazine (25 mg and 75 mg) on primary arousal and on a compensatory mechanism (motivation) (Hartley *et al*, 1978). Their findings suggested that chlorpromazine impaired arousal but not drive or motivation. Hartley (1983) concluded that chlorpromazine impaired and amphetamine improved performance on tests of vigilance, and also in RT tests of attention which were paced but not those which were unpaced. This was in contrast to the global impairments caused by non-specific sedatives including alcohol. Accordingly it was proposed that the catecholamine system was principally involved in stimulus selection as opposed to response speed.

An unexpected beneficial effect of chlorpromazine on information processing was reported by Stone *et al* (1969), who compared the effects of chlorpromazine (50 mg), scopolamine (0.6 mg), pentobarbital (100 mg) and placebo on a short-term memory task. Performance on this test, which was administered to 40 volunteers in a group, 45 minutes after drug ingestion, appeared to be improved by chlorpromazine, whereas the barbiturate, scopolamine and placebo were indistinguishable. However, the timing of the test was not likely to have detected a true chlorpromazine effect and no dose–response relationship was demonstrated.

(iv) Comparisons between phenothiazines

Comparisons of the effects of different phenothiazine derivatives were made by DiMascio *et al* (1963*a*,*b*). In that series of studies the effects of four single doses of a sedative, low-potency phenothiazine (chlorpromazine, 25, 50, 100 and 200 mg), a sedative, ineffective phenothiazine (promethazine, 25, 50, 100 and 200 mg), and two non-sedative piperazine phenothiazines (perphenazine and trifluoperazine, 2, 4, 8 and 16 mg) were compared in 36 student volunteers. Chlorpromazine and promethazine invariably impaired psychomotor performance and speed (but not accuracy) of cognitive function, while perphenazine and trifluoperazine, even at the highest dose, either had no effect on these measures (e.g. DSST) or actually improved performance (e.g. tapping speed, pursuit-rotor time, symbol copying and serial addition).

Furthermore, when the effects of alcohol and phenothiazine combinations were studied by Milner & Landauer (1971), both chlorpromazine and thioridazine in doses of 1.0 mg/kg were shown to impair performance on tracking, pursuit-rotor and simulated driving tests. An additive interaction with alcohol was found with both drugs, and potentiation occurred with chlorpromazine and alcohol on the pursuit-rotor test.

More recent studies have employed much lower doses, but psychomotor effects then become more difficult to detect. Thus neither single 25 mg doses of chlorpromazine nor chronic low-dose treatment (20 mg t.i.d. for two weeks) affected memory, learning or psychomotor performance in 20 normal volunteers (Liljequist *et al*, 1975, 1978). No effect on memory or learning was detected in a chronic low-dose study with thioridazine (10 mg t.i.d. for two weeks) in 20 volunteers (Liljequist *et al*, 1975), nor were any psychomotor effects detected after thioridazine 10 mg t.i.d. for one week followed by 20 mg t.i.d. for a second week in 20 volunteers

(Saario, 1976). Single 50 mg doses of thioridazine, however, were shown to impair both subjective alertness and psychomotor performance in two studies using eight volunteers (Szabadi *et al*, 1980; Theofilopoulos, 1984), and doses of 1.5 mg/kg in a study with only four volunteers (Meyer *et al*, 1983). It may have been that tolerance developed to chlorpromazine and thioridazine in the studies employing chronic low doses, but since impairments to single low doses (less than 50 mg) have not been demonstrated, this remains speculative.

(b) Haloperidol

Four studies that focused on subjective effects and provided no objective measures of performance reported negative effects of haloperidol (in doses ranging from 0.25 mg intramuscularly to 8.75 mg intravenously), particularly akathisia and dysphoria (Pfeiffer *et al*, 1968; Belmaker & Wald, 1977; Anderson *et al*, 1981; Magliozzi *et al*, 1985). One of these (Anderson *et al*, 1981) reported akathisia lasting from 36 hours to five days and dysphoria for up to six weeks in one subject, after 5 mg orally. In another, such high doses of oral (0.5 mg/kg) or intravenous (0.125 mg/kg) haloperidol were given that the subjects were often unable to complete the tasks (Magliozzi *et al*, 1985).

However, several systematic studies with low doses of haloperidol in normal volunteers have yielded interesting results, with the suggestion of improvement in performance on some tests. Thus, in a study of 138 student volunteers, James & James (1973) reported that DSST performance improved with haloperidol (0.33 mg t.i.d. for 24 hours before testing) compared with chlordiazepoxide (10 mg t.i.d. for 24 hours), but their data suggest that this dose of haloperidol was indistinguishable from placebo. In fact higher doses of haloperidol (4 mg and 10 mg) have recently been shown to cause dose-dependent decreases in DSST scores, with peak effects between four and six hours, which coincide with elevations in prolactin (Magliozzi *et al*, 1989). Parrott & Hindmarch (1975) demonstrated in six volunteers that, while chlorpromazine (50 mg) impaired both arousal (as measured by CFFT) and thinking time (as measured by response latency in a CRT test), haloperidol (1 mg) impaired arousal but improved latency two hours after dosing. The haloperidol effect on latency, however, was again similar to that of placebo. Thus it seems more likely that low doses of haloperidol (1 mg) have not been detected by the DSST and CRT tests used by James & James (1973) and Parrott & Hindmarch (1975), rather than causing any improvement in performance. Janke & Debus (1972), however, maintain that single low doses (1 mg and 2 mg) of haloperidol and pimozide can either improve or impair performance depending upon the personality of the subjects and the work-load or difficulty of the experimental test. They studied 13 emotionally unstable subjects but did not make direct comparisons with stable subjects. A high work-load involved complex serial additions of high subjective difficulty before psychomotor testing. They found subjective activation and improved performance on finger tapping speed with haloperidol (2 mg) and a low work-load for neurotic subjects, but these effects were reversed with a high work-load. Pimozide (2 mg) had similar effects with a low work-load, but no negative effects with a high work-load. Cognitive performance (word fluency) improved with pimozide with a low work-load, but was unaffected by haloperidol. These data were interpreted as showing that with a low work-load the tranquillising properties of neuroleptics (on the limbic system) had beneficial effects in emotionally unstable subjects, but that under conditions of greater difficulty their sedative properties (on the arousal of the reticular formation) interfered with performance and hindered their beneficial effects. It is uncertain, however, whether the same or different effects would occur in emotionally stable subjects. This two-component model of neuroleptic drug action was an extension of Eysenck's (1957) ideas on optimal arousal, and incorporated both the motivational and difficulty aspects of the Yerkes–Dodson Law (Broadhurst, 1957).

Saletu *et al* (1983*a*,*b*) compared single doses of haloperidol (2 mg) with an atypical neuroleptic (zetidoline) and placebo in ten normal volunteers. Statistically significant activating effects were found for haloperidol on simple and complex reaction times (at four hours), and on mood and time estimation (at two hours). However, the peak haloperidol effect as measured by the rise in prolactin levels and changes in EEG parameters was at six hours, by which time attention and concentration (as measured by a letter cancellation task) and CFFT were impaired by the haloperidol. Likewise, using a recently developed rapid-information-processing task, McClelland *et al* (1987) and Cooper *et al* (1989) found significant impairments at four hours after single doses of haloperidol (3 mg) in 12 volunteers. Saarialho-Kere (1988), however, failed to find any effect of four repeated low doses of haloperidol (0.5 mg) in 12 volunteers on a number of conventional measures of psychomotor performance, including CFFT, CRT, Maddox wing, attention and tracking, at up to four hours. These doses of haloperidol were, nevertheless, adequate to cause elevated prolactin levels. Similarly, Frey *et al* (1989) reported that, although 0.75 mg haloperidol intravenously caused sedation and elevations in prolactin in six volunteers, no effect on CFFT or pharmaco-EEG measures were detected.

(c) Droperidol

Recently more sophisticated and ingenious paradigms have been used to develop more selective tests of attention (divided and focused) and information processing. Using a difficult dichotic monitoring task, Clark *et al* (1987) found that intravenous clonidine (200 μg) caused decreases in both attention capacity and processing speed, which were related to changes in the level of arousal. Although intravenous droperidol (15 μg/kg) had similar effects on attentional performance, it did not alter subjective activation or arousal. Effects on the switching of attention were measured using the Posner covert orientation of attention test (Clark *et al*, 1989). In this task, responses are measured to targets 25° lateral to the left or right while the eyes remain fixed to a central point. The targets are preceded by either neutral, valid (true) or invalid (false) cues and the 'benefits' of valid cuing, or the 'costs' of invalid cuing, can be measured. Although both droperidol (15 μg/kg i.v.) and clonidine (200 μg i.v.) increased overall response times, they were found to reduce the cost of invalid cuing. There was no change in the effects of neutral or valid cuing. It was therefore argued that both drugs improved attentional processing by facilitating the shifting of attention. These effects were contrasted with those of amphetamine and lesions of the inferior parietal lobe, which respectively enhance attention during vigilance tasks and impair the disengagement of attention. These studies need to be repeated to see if the effects are peculiar to drugs affecting catecholamine systems, and to demonstrate dose-dependent effects. The potential implication for schizophrenia is that neuroleptics may act both by decreasing general attention and facilitating attentional shift, in a state of excessively fixed or focused attention.

(d) Flupenthixol

Rösler *et al* (1985) reported that flupenthixol (2 mg/day for four days) impaired reaction time and information processing, as measured by a pharmaco-EEG study of endogenous event-related potentials (CNV and P300 waves) in 54 healthy male volunteers. They also examined the interaction between the effects of flupenthixol (up to 2 mg daily for four days) and personality variables on 15 different psychomotor and cognitive tests in 107 normal subjects, using multivariate analysis of variance, in a further placebo-controlled study (Rösler *et al*, 1986). They found that, while stable personalities performed better than unstable personalities overall, there was no interaction between personality and drug effects. This suggests that the findings of Janke & Debus (1972) (see above) might apply to both emotionally stable and unstable personalities. Rösler *et al* (1986) found that flupenthixol had predominantly detrimental effects on perceptual and psychomotor function, although isolated improvements were noted in some tests, with neurotics improving more than stable personalities in some of these and vice versa in others. This was interpreted as changes in arousal being either optimal or sub-optimal according to different personality traits and test requirements. In contrast to these subchronic dosing studies, Mattila *et al* (1988*a*) were unable to detect any effect of single 1 mg or 2 mg doses of flupenthixol on a range o psychomotor and cognitive function tests, apart from transient impairments on two tests (tapping and tracking) after 2 mg.

(e) Atypical neuroleptics

Sulpiride, either in single doses of 100 mg (Bartfa & Wiesel, 1986) or chronic low doses of 50 mg t.i.d for two weeks (Liljequist *et al*, 1975), has not been shown to cause any psychomotor or cognitive impairment in conventional tests. Similarly, von Aschof *et al* (1974), using electro-oculogram nystagmography found that sulpiride (300 mg daily for one week) was devoid of any effect on the velocity or latency o saccadic eye movements, and, in fact, saccadic accuracy was slightly improved. Remoxipride however, in single doses of 30 mg and 60 mg, does cause measurable impairments in CFFT, CRT latency and continuous attention (Fagan *et al* 1988). Performance on a DSST and a tracking test were also impaired by 100 mg doses of remoxipride (Mattila *et al*, 1988*b*). Interestingly, this new atypical neuroleptic is a selective post-synaptic D_2 antagonist whereas sulpiride is a selective pre-synaptic D antagonist and the early activating effects of low doses of haloperidol have been posited to be related to a pre-synaptic effect (Saletu *et al*, 1983*b*).

The low-potency dopamine antagonist zetidoline (DL-308) has been studied by three groups. In a study of single 10 mg and 20 mg doses given to eight volunteers, Szabadi *et al* (1980) found 20 mg to be more sedative than 50 mg thioridazine but with weaker α-adrenoceptor-antagonist and anticholinergic effects. Dose-dependent subjective and objective impairments were found on visual analogue scales symbol copying, letter cancellation, DSST, finger tapping and wire-maze tracing. Similar findings were reported by Herbert *et al* (1983) in a study of eight volunteers in which more prolonged tests were used (lasting five to ten minutes each) (visual search, Baddeley logical reasoning and choice serial reaction time) and which were thought to detect more cognitive aspects of function. Saletu *et al* (1983*a,b*) also reported impairments with 10–40 mg doses of zetidoline on mood and psychomotor function in ten volunteers. In addition, their quantitative EEG analyses showed that zetidoline decreased alpha activity and increased both theta and beta activity, a pattern similar to that subsequently reported with fluperlapine (Saletu *et al*, 1986), clozapine, tricyclic antidepressants and anticholinergic drugs (Saletu *et al*, 1987).

Saletu *et al* (1987) also studied the effects of single doses of clozapine (50 mg) and zotepine (25 mg to 100 mg), a new clozapine-like compound with antidopaminergic, adrenolytic, antiserotonergic and anticholinergic properties, in 15 volunteers. Zotepine caused similar impairments to clozapine on tests of cognition, psychomotor performance and mood, but with a later onset and a more prolonged action. Its EEG profile was similar to that of chlorpromazine, with decreased alpha and beta activity and increased theta and delta activity.

Similar effects of acute doses of fluperlapine on cognitive and psychomotor function in schizophrenic patients were also reported by this Viennese group, as previously mentioned above (Saletu *et al*, 1986).

Discussion

A major difficulty in comparing the studies reported above is the great variety of tests and methodologies employed. Not surprisingly, therefore, there is little consistency in the reported results, and simple generalisations risk being misleading. In clinical studies the type and severity of illness has not always been clear, and information on the type of drug, dose and length of exposure has sometimes been very general and vague. While volunteer studies provide more precise drug information, variables such as age, sex, motivation and personality have not always been considered. Generally, the clinical studies have been strong on the assessment of cognition but weak on pharmacological methodology, and vice versa with volunteer studies.

Recurring problems with conventional tests of psychomotor and cognitive function are the effects of practice and fatigue; motivation and/or co-operation; attention, both voluntary and involuntary; inter- and intra-individual variation; differences in sensitivity between tests of similar functions; and lack of selectivity of the tests for a particular function. Some of these can be avoided by improved experimental design and rigour, for example the effects of practice and fatigue, but some earlier studies were patently flawed in this respect. No test or design, however, has overcome the problems of wide inter- and intrasubject variability. The only way of dealing with this is to study as large a number of subjects as possible. It would appear that at least 8–12 subjects are required to detect the effects of 50 mg of chlorpromazine or thioridazine or 2 mg of haloperidol reliably with conventional psychomotor tests.

Motivation is difficult to estimate and control, and has often been held to account for the differences in performance between patients and normal volunteers. Attempts can be made to estimate this, either by using rating scales (Spohn *et al*, 1985) or by having simpler 'control' tests, such as number matching, included in the test battery (Berman *et al*, 1986). Spohn *et al* (1985) carried out stepwise multiple-regression analyses of the effect of a number of clinical variables, including motivation ('general deficit functioning'), tardive dyskinesia and drug dose, on both psychomotor and information-processing performance and on psychophysiological abnormalities, in 84 chronic schizophrenics. They found that motivation accounted for a significant amount of the variation in reaction time, span of apprehension and basal skin conductance. Such a rigorous analysis is rare, and it also showed that high drug doses and tardive dyskinesia were important sources of error in patient studies. Their findings indicate that in their earlier study (Spohn *et al*, 1977), which did not control for such interactions, a negative effect of neuroleptics on attention and cognition had been obscured by improvements due to clinical recovery. Motivation itself, however, would not appear to be impaired by neuroleptics (Hartley *et al*, 1978).

None of the tests so far developed has been shown to be selective for a particular psychomotor or cognitive function. Neither has the sensitivity always been adequate to detect the subtle effects of neuroleptics. The CPT was thought to be particularly useful in schizophrenic patients (Kornetsky, 1972) and to distinguish between phenothiazines and barbiturates (Mirsky & Kornetsky, 1964; Kornetsky & Orzack, 1964), but later studies have produced varied results (Medalia *et al*, 1988). It now seems clear that a general decrease in brain-stem arousal does not account for the beneficial effect of neuroleptics (Janke & Debus, 1972; Hartley, 1983). The WCST has been thought to be a relatively selective measure of frontal lobe function (Berman *et al*, 1986), but this, too, has been challenged (Frith, 1987). Maze tests of frontal lobe function do not seem to be reliable, but show trends towards impairment by neuroleptics (Porteus, 1957; Medalia *et al*, 1988; Cleghorn *et al*, 1990). Moreover, the duration of the test rather than its inferred 'cognitive load' or functional localisation may be what determines sensitivity to drug effects, since, for instance: (a) a performance on the DSST was more impaired by barbiturates than chlorpromazine, whereas the difference in performance on the longer CPT was much less (Mirsky & Kornetsky, 1964); (b) Szabadi *et al* (1980) found digit cancellation to be more sensitive than the DSST to the effects of thioridazine and zetidoline; and (c) the choice serial reaction time (ten minutes) detected the effects of oxazepam (30 mg) and zetidoline (10 mg and 20 mg), whereas the Baddeley logical reasoning test (five minutes) only detected the effects of 20 mg zetidoline and a five-minute visual search test was not sensitive to either drug (Herbert *et al*, 1983).

More precise tests of information processing such as the backward visual-masking task (Braff & Saccuzzo, 1982), the rapid information-processing test (McClelland *et al*, 1987), or a dichotic monitoring task (Clark *et al*, 1987) have produced promising results. The covert attentional orientation test (Clark *et al*, 1989) also merits further study, particularly since it offers an explanation of how drugs that produce negative effects in normals have beneficial effects in schizophrenics. Nevertheless, the beneficial effects of droperidol on attentional shift, as measured by this test, are the converse of those of haloperidol as measured by card-sorting and word-fluency tests (Berger *et al*, 1989).

Conclusions

No single hypothesis has emerged for the mechanism by which neuroleptics produce their beneficial effects in schizophrenia. Speculations about drug action tend to derive, in part, from theories of the pathophysiology of the condition, and vice versa. Three ideas are suggested by the available data reviewed here.

(a) Neuroleptics primarily inhibit mesolimbic dopamine activity and subcortical psychomotor functions, leaving higher cognitive function largely intact. Thus improvement in attention is facilitated by indirect mechanisms. This is consistent with the Jacksonian view of positive psychotic symptoms being 'release' phenomena.

(b) Normalisation of asymmetrical temporohippocampal function may occur.

(c) Improved attentional processing, particularly facilitation of the shifting of attention, may occur in spite of an overall slowing of response speed.

The way forward appears to be to continue to develop improved paradigms of attentional processes and information processing. The effect of the new, more selective drugs on these may elucidate the physiological mode of action of neuroleptics, the role of dopamine in attentional processes (Matthysse, 1978), and the pathophysiological basis of abnormal cognition in schizophrenia.

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Review Article

The Similarities and Dissimilarities Between Community and Clinic Cases of Depression

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It has been hypothesised that community cases of depression compared with clinic cases: (a) have fewer symptoms; (b) have milder symptoms; (c) have different symptoms; (d) are of shorter duration; (e) are less incapacitating; (f) are more environmentally and less biologically caused; and (g) respond better to psychological than pharmacological intervention. A review of the literature indicates that some of these hypotheses have not been tested, others have not been adequately tested, and none has been unequivocally confirmed. Further investigations of these important issues are required.

There have been many community investigations into the psychosocial factors associated with depression (e.g. Brown & Harris, 1978*a*; Bebbington *et al*, 1981*a*,*b*; Costello, 1982; Surtees *et al*, 1986). In these studies, random samples of the community have been interviewed, and standardised psychiatric interviews, ratings and computational procedures have been used to identify individuals who satisfy one or other set of criteria for depression. The usefulness of these studies for understanding the nature of depressed patients attending clinics as out-patients or in-patients has been questioned. For instance, Thase *et al* (1985) wrote that: "it is difficult, if not impossible, to generalise findings from such investigations to the samples of major depressives studied in biological research settings" (p. 879). Similar concerns have been expressed by Tennant & Bebbington (1978), Richman & Barry (1985), Akiskal (1987), and Snaith (1987). Counterarguments have been presented by Brown & Harris (1978*b*, 1982, 1986), relying mostly on their own data. The present review is broader in its coverage of the relevant literature and discusses some issues not discussed in the articles by Brown & Harris.

It has been hypothesised that there are one or more of seven differences between the depressions identified in the community and those found in clinics. Community depressions compared with clinic depressions: (a) have fewer symptoms; (b) have less severe symptoms; (c) have different symptoms; (d) are of shorter duration; (e) are less incapacitating; (f) are more environmentally and less biologically caused; and (g) are more responsive to psychological and less responsive to pharmacological intervention. This review examines the degree to which there is empirical support for these hypothesised differences. The studies selected have all used standardised psychiatric interviews as well as established symptom rating procedures and diagnostic algorithms.

Number of symptoms

Two studies have examined the total number of symptoms in their cases of depression. Brown *et al* (1985), using the Present State Examination (PSE; Wing *et al*, 1974), interviewed a community sample of 363 working-class women at two points in time 12 months apart. The first interview indicated that the 64 women who had suffered from depression during the previous 12 months had an average total of 19 PSE symptoms. Wing (1976) reported an average total PSE score of 15 for 19 cases of depression identified in a random community sample. The means for 55 depressed in-patients and 24 depressed out-patients reported in Wing (1976) were 26 and 24 respectively.

These data do appear to support the hypothesis that clinic cases of depression have more symptoms than community cases. However, the short form of the PSE, which includes 40 items, mostly symptoms, was used in the community studies and the long form, which includes 140 items, mostly symptoms, was used in the clinic studies. Furthermore, there is evidence (e.g. Shapiro *et al*, 1984) that individuals with more than one diagnosable psychiatric condition are more likely to seek referral or be referred to psychiatric clinics. Both of these differences might be expected to result in a higher total PSE score in the clinic samples. On the other hand, cases identified in community samples do not always meet criteria for only one diagnosis. Brown *et al*'s data (1985, p. 617) suggest that, although there were 64 depressed women, only 29 were 'pure' depressives – the remaining 35 of the women also had an anxiety condition. Examination of the data for the 29 'pure' cases, made available by Brown (personal communication, 1988), indicated that two cases also had substance abuse disorders and a further two had contacted out-patient psychiatric services. Thus, there were only 25 cases who were both true community cases *and* 'pure' depressives. The average total number of PSE symptoms for this group was 16.

The question of whether community depressions differ from clinic depressions in the number of depressive symptoms can only be answered satisfactorily by investigating 'pure' cases of community and clinic depressions and by counting only those symptoms that are considered to define, in part, the syndromes of depression.

Severity of symptoms

Data comparing the severity of symptoms in community and clinic cases of depression do not appear to have been reported. In any case, such data would have to be cautiously interpreted. If it were shown that clinic cases had more severe symptoms this might be because patients in clinic samples are usually interviewed on admission, when they are at or near the peak of their disorders. Brown & Harris (1978*a*) measured overall severity of illness in 41 depressed out-patients and 73 depressed in-patients using a five-point scale (1, marked; 5, mild). There was little change in severity score from onset of illness (mean 2.93) to admission (mean 2.54) for out-patients but the mean severity score for in-patients changed from 2.73 at onset to 1.86 at admission. When assessments in random community samples are based on the month before interview, some of the cases, although meeting diagnostic criteria, may not be close to the peak of their disturbances. When such assessments are made for onsets earlier than one month before interview, the passage of time may affect the recall of symptoms. Aneshensel *et al* (1987) found that individuals tended to fail to report documented past episodes of depression when they were currently asymptomatic or when current life circumstances were positive or had improved.

Nature of symptoms

Only one study seems to have provided data concerning differences between community and clinic cases in the nature of their depressive symptoms. In this study, different aspects of which were reported in Wing *et al* (1981) and Hurry & Sturt (1981), PSE symptoms data were processed through the Index of Definition (ID) and CATEGO computer programs (Wing *et al*, 1974, 1978). The ID program allocates each individual to one of eight levels, which indicates the degree of confidence that sufficient symptoms are present to allow a clinical classification. Level five represents the threshold category. Individuals at levels six, seven and eight are considered to be definite cases. The CATEGO program allocates each individual with an ID of five or above to one of a number of categories which are approximately equivalent to the diagnostic categories of ICD–9 (World Health Organization, 1978). Calculations based on the tables in Wing *et al* (1981) and Hurry & Sturt (1981) indicate that of 81 depressed in-patients 19% were in CATEGO class D (i.e. they suffered from delusions and/or hallucinations); 43% were in class R (characterised by symptoms such as motor retardation, pathological guilt and self-deprecation); and 38% were in class N (neurotic depression). The distribution of cases for the 45 depressed out-patients was: 4% in class D; 27% in class R; and 69% in class N. The comparable figures for the 35 depressed community cases were 6%, 24% and 71%, respectively. Thus, the distribution of cases is not different for the out-patients and community cases, but a greater proportion of the in-patients are in classes D and R. Nevertheless, the data obtained from the R and N community depressions (95% of the cases) might be generalisable to the class R and N depressed in-patients (81% of the cases).

Calculations based on the data in Wing *et al* (1981; Table 6.6) indicate that 40% of 35 community cases of depression compared with 82% of 45 depressed out-patients had above-threshold IDs (i.e. ID$\geqslant$6), (insufficient data were presented to permit similar calculations for the depressed in-patients.) The difficulty presented by such a difference in IDs is that the source of this is unknown. The ID is more than simply a sum of symptoms reported and the rules for determining its level operate both on total PSE scores and on type, severity and combination of symptoms present (Wing & Sturt, 1978).

Validity of data from lay interviewers

The studies reviewed so far have suggested that clinic cases of depression might have more symptoms and more severe symptoms. However, there is no clear indication that community depressions are significantly different from clinic depressions, in relation to the nature of the depressive symptoms *per se*. Moreover, most of the community data have been collected by lay interviewers and the validity of such data, using psychiatrists' diagnoses as the standard, has not been firmly established. It is sometimes suspected that lay people, using normal behaviour as their standard, will tend to over-rate symptoms, and Copeland *et al* (1975) found that non-psychiatrists (psychology students, nurses and general practitioners) gave higher symptom ratings than psychiatrists when viewing a videotaped interview with a psychiatric patient. However, this is not

TABLE I

Agreement between ratings of lay interviewers and psychiatrists on presence or absence of three PSE syndromes of depression in 28 women (data abstracted from Table 1 of Wing et al*; 1977)*

PSE syndrome	*Overall agreement*	*Agreement: symptom absent*	*Agreement: symptom present*	*Kappa*
Simple depression	0.93	0.90	0.80	0.64
Special features of depression	0.96	0.96	0.67	0.78
Other symptoms of depression	0.93	0.89	0.83	0.85

surprising since the non-psychiatrists had received no training whatsoever in the ratings of symptoms. Such data do not cast doubt on symptom data obtained by *adequately trained* lay interviewers.

In general, except for one study (see below), all the available studies do not permit any firm conclusion concerning the validity of community data obtained by lay interviewers using standardised psychiatric interviews such as the PSE and using the results obtained by psychiatrists as the gold standard. This is because of one or other of the following methodological problems: (a) the lay people received no training in the interview and rating procedures or less training than the psychiatrists (Copeland *et al*, 1975; Cooper *et al*, 1977; sections of Wing *et al*, 1977); Sturt *et al*, 1981; (b) the assessment procedure used in one of the studies (Copeland *et al*, 1975) was the Lorr in-patient multi-dimensional psychiatric scale which has not been used in community studies; (c) the ratings compared were based on interviews at relatively long intervals apart (sections of Wing *et al*, 1977) with no controlling for real changes in symptoms between the interviews or for the possible effects of repeat interviews; and (d) the subject sample was a clinic rather than community sample (Copeland *et al*, 1975; Cooper *et al*, 1977; Wing *et al*, 1977).

In the one study that was free of these methodological problems, interviews of a random sample of 28 women in the community were conducted by four lay people who had received the full standard training on the PSE (sections of Wing *et al*, 1977). One of three psychiatrists listened to the audiotapes of each interview. The correlation for the total PSE scores based on the ratings of the lay people and the psychiatrists was 0.96. The data on the degree of agreement for three different syndromes of depression produced by the CATEGO program are presented in Table I. These data suggest that there is considerable agreement between the ratings of lay people and psychiatrists. However, the sample was small, all the interviews were conducted by the lay interviewers, and the issue is in need of more thorough investigation.

Since the validity of data provided by lay interviewers, using open-ended instruments such as the PSE, has not been firmly established, some psychopathologists may advocate a different approach, using a more structured instrument such as the Diagnostic Interview Schedule (DIS; Robins *et al*, 1981) which does not require the interviewer to make any ratings as the symptom codes follow directly from the interviewee's responses. However, two recent studies cast doubt on such a solution.

In a random community sample of subjects to whom the DIS had been administered by trained lay people, 358 were selected for interview by psychiatrists so that as many as possible DSM-III diagnoses (American Psychiatric Association, 1980) were covered and so that a sample of those without a DSM-III diagnosis were included. The psychiatrists then administered a second set of DISs and were able to supplement these with any probes they felt were necessary to obtain adequate information for diagnoses. The psychiatrists' clinical diagnoses were based on DSM-III criteria. The mean time between the first and second interview was six weeks. Since lifetime rather than current diagnoses were derived from the data, this interval probably did not introduce large systematic errors due to a change in the subjects' conditions. However, there may have been some effects of repeated testing. The data for the lifetime diagnoses of major depression are presented in Table II. It can be seen that, taking the psychiatrists' diagnoses as the standard, the sensitivity of the DIS administered by the lay interviewers was only 0.42.

Anthony *et al* (1985) presented data for 810 community subjects who had been administered the DIS by a lay interviewer followed by an interview with a psychiatrist using a PSE augmented to cover all of the DSM-III criteria. The individuals were selected from a larger random community sample in the same manner as that used by Helzer *et al*

TABLE II

Lifetime diagnosis of major depression (from Heltzer et al, *1985; Table 5) and one-month diagnosis of major depression (from Anthony* et al, *1985; Table 3); both obtained for community samples of subjects (*n = *358 and 810, respectively) by lay interviewers' administration of the DIS and by psychiatrists' clinical judgements based on the administration of a modified DIS*

Psychiatrists' diagnosis	*Lay interviewers' DIS results*			
	*Lifetime diagnosis (*n = *358)*		*One-month diagnosis (*n = *810)*	
	Major depression	*No major depression*	*Major depression*	*No major depression*
Major depression	42	59	13	9
No major depression	15	242	48	740

(1985). Two-thirds of the 'clinical reappraisal' examinations were completed within three weeks of the DIS interview, 75% within four weeks and 93% within 90 days. Of the 810 subjects reinterviewed, 519 had a one-month DIS-DSM-III diagnosis based on the first interview and the remaining 291 had no diagnosis. The results for a one-month diagnosis of major depression are also presented in Table II. The misclassification rate of 7% is low. But the sensitivity of the DIS administered by lay interviewers is only 0.59 and the positive predictive value of the DIS diagnoses is only 0.21. Once again, some of the disagreement may be due to the lapse of time between the two interviews and to the effects of repeated assessment.

Duration of condition

Brown & Harris (1986) noted that in all of their community studies there were as many women with episodes of depression which lasted 12 months or more as those with new episodes that had their onsets in the year before interview. Other studies have reported a lower proportion of chronic depressions. For instance Costello (1982) found a 17% rate. However, because of the difficulties of studying the aetiology of chronic conditions (Brown & Harris, 1986; Depue & Monroe, 1986), community investigations of the psychosocial correlates of depression have concentrated on episodes that have had their onset during the period covered by the interview. Only one of the studies obtained data on the duration of these episodes: when 32 women with onsets of depression during the 12 months before their first interview were interviewed again 12 months later, it was found that 47% of the depressions had lasted for at least six months (Brown *et al*, 1985). However, examination of the data made available by Brown (personal communication, 1988) for the 25 'pure' cases of depression who had not had contact with out-patient services, indicated that 13 (52%) had been depressed for over a year. The duration of illness for the remaining 12 'pure' cases ranged from two to nine months (mean 5.5). Hollon's (1986) review of studies conducted before the introduction of antidepressants indicated that the average duration of untreated episodes of depression was three to six months for out-patients and six to nine months for in-patients.

The above data suggest that the duration of depression in community cases is comparable with that found in clinic cases. However, Brown & Harris (1978*b*) suggested that to use duration as a criterion for distinguishing true clinic depression from community depression is conceptually suspect until empirical study can show that duration is an indication of some other difference between the conditions. They also noted that, even if community depressions do not generally last as long as clinic depressions, it is obviously important to include short-lived conditions in our investigations if we are to understand more about the factors that help perpetuate depressive disorders.

Social incapacitation

Hurry & Sturt (1981) found that 18% of 35 community cases of depression had scores of 0 on the Social Performance Schedule (Creer & Wing, 1974); the scores were between 1 and 10 for 27%; between 11 and 20 for 18%; between 21 and 30 for 23%; between 31 and 100 for 15%, with higher scores indicating greater social incapacitation. The comparable figures for 45 depressed out-patients were 4%, 11%, 22%, 18%, and 44% respectively. These data suggest that clinic cases are more socially incapacitated than community cases, but it would be premature to conclude that this difference was a direct result of a difference in the severity or nature of their depressions. Referral to clinics is influenced by a number of factors including anxiety, alcoholism, and drug addiction, as well as civil status (Brown & Harris, 1978*a*; Brown *et al*, 1985; Mechanic, 1972; Goldberg & Huxley, 1980; Shapiro *et al*, 1984). These factors could well influence social incapacitation. Hurry & Sturt (1981) indicated that when individuals with similar levels of symptoms were compared, those with a poor level of social performance, as measured by the Social Performance Schedule, were more likely to contact or be referred to out-patient services.

Environment versus biology

To support their argument that community depressions are more environmentally caused than are clinic depressions, Bebbington *et al* (1981*b*) reported data from an investigation of a random community sample of 310 subjects, interviewed on two occasions using the PSE and the Life Events and Difficulties Schedule (LEDS; Brown, 1978) and 74 psychiatric out-patients. The ID and CATEGO programs were applied to the PSE data. Those who were at level five or over on the ID at the first or second interview were defined as cases. However, since the authors doubted the reliability of some of the data from the first interview, they used two definitions to identify 'cases': the 'broad definition' included cases identified at both interviews and the 'narrow definition' included only the cases identified at the second interview. Whereas 47% of 19 female community cases ('narrow definition') had had a severe life event in the previous three months, only 18% of 33 female out-patients had had such an event. They concluded that these data and similar data obtained for males supported their claim that "disorders seen in patients differ from (at any rate, acute) disorders seen in the community in their relationship to adversity. This difference supports the suggestion that disease theories are more likely to be required to explain the occurrence of the more severe affective disorders, whilst less severe disorders often have a ready explication as understandable and unmysterious responses to adversity" (p. 364). However, Brown & Harris (1982) reviewed seven studies of depressive in-patients and out-patients and noted that 44–73% (mean 56%) of such patients reported a severe event before onset. Furthermore, Brown *et al* (1985) proposed that the low rate of stressors in the study by Bebbington *et al* (1981) could be accounted for by three aspects of their design: (a) counting events over only the three months before onset; (b) the exclusion of 'possibly independent' events (i.e. events over which the patients might have had some control) and (c) the inclusion, in their small sample of patients, of cases of anxiety without depression.

The belief that community cases may be more caused by environmental factors than clinic cases rests on: (a) the observation that a greater proportion of clinic patients than community cases are of an endogenous or melancholic nature; and (b) the continued acceptance of the hypothesis that endogenous depressions are less related to environmental factors than non-endogenous depressions. As Bebbington *et al* (1988) noted, there have been a number of multivariate studies that have found that adversity discriminates neurotic from endogenous depressives. However, two methodological problems suggest that the findings may be an artefact: (a) comparisons were made between endogenous and neurotic groups that had been diagnosed by clinicians, who usually make the distinction on grounds of both characteristic symptoms and the presence or absence of potential psychosocial stressors; and (b) age was not statistically controlled, which presents a problem because depression in older people is more likely to be of an endogenous type and life events are rarer in older people. When Paykel (1971) did control for age in a multivariate study, the discriminant power of adversity was lost.

There are now a considerable number of studies reporting no differences between endogenous and non-endogenous types of depression in the rates of severe events. For instance, Benjaminsen (1981), adopting Paykel *et al*'s (1971) classification of types of events, found no difference in rates of 'severely upsetting events' for neurotic depressives (mean 83%) and non-neurotic depressives (mean 90%), nor in the rates of 'undesirable events' (means of 91% and 84%, respectively) or the rates of 'severe losses' (means of 65% and 58% respectively).

Bebbington *et al* (1988) found that 38% of 63 psychiatric out-patients and in-patients classified by CATEGO as endogenous depressives and 39% of 61 neurotic depressives had a severe event during the three months before onset. When chronic difficulties were included with events to constitute the variable 'provoking agent', 48% of the endogenous and 49% of the neurotic patients had a provoking agent before onset of illness. Paykel *et al* (1984) found that the relationship between the absence of life stress and the presence of endogenous symptoms was weak, since only 18 of 105 correlations reached the 5% level of significance. Calloway *et al* (1984*a*,*b*) found that depressed patients with the clearest signs of biological involvement (i.e. non-suppression on the dexamethasone suppression test (DST) and blunted response of thyroid-stimulating hormone (TSH) to thyrotropin-releasing hormone (TRH)) were as likely to have had an antecedent stressor as the remaining depressed patients. In fact, patients with blunted TSH responses and a high free thyroxine index (FTI) were more likely to report long-term environmental difficulties than patients with blunted responses and normal FTI values.

Bebbington (1984) suggested that "A biological basis for depressive illness is best sought through genetic studies, as biological markers such as the dexamethasone suppression test may merely be correlates of symptoms rather than correlates of the causes of symptoms" (p. 71).

There do not appear to be any studies directly comparing the heritability of community and clinic cases of depression. Of indirect relevance, however, are Torgersen's (1986) findings based on 151 index twins, all of whom were depressed in-patients, and their co-twins – that hereditary factors were important in the aetiology of depression. The frequency of major depression in monozygotic (MZ) co-twins of patients with major depression was 27% compared with 12% among dizygotic (DZ) co-twins. This MZ/DZ difference was not statistically significant, but when all affective disorders in the co-twins were considered, the concordance rate for the MZ twin pairs was 45% compared with 22% in the DZ pairs, and this difference was statistically significant. Of greater importance, however, was Torgersen's finding that the concordance rates for subtypes of major depression (with and without melancholic features) and with and without an antecedent severe event) for the MZ and DZ twins were similar.

If indeed environmentally caused depressions do not have significant biological determinants, then one might expect little in the way of a family history of depression in those who have a severe event preceding onset of depression. However, McGuffin *et al* (1987, 1988) noted that the difference between the 21% lifetime rate of depression in the relatives of probands whose onset of depression followed life events or chronic difficulties and the 13% rate in the relatives of probands whose onset was not associated with adversity is statistically insignificant. Furthermore, the trend, although insignificant, is in the opposite direction to that required by the hypothesis.

Response to pharmacological intervention

Akiskal (1987) argued that measurement of pharmacological response is a useful approach to distinguishing between, for example, an adaptive response to loss and clinical depression. "Morbid depression is often alleviated by tricyclic antidepressants, whereas ordinary sadness is unaffected. By contrast, stimulants with immediate mood-elevating properties have no long-term benefit in clinical depression" (pp. 64–65).

There do not seem to have been any studies which have compared the response of community depressives to psychological and pharmacological intervention, and only one study (Nandi *et al*, 1976) has investigated the response of community cases to pharmacological treatment. In this, the 17 cases identified in the community responded as well to imipramine as 20 cases of depression treated in a clinic. The authors noted that there was no significant difference between the two groups in the mean score on the Hamilton Rating Scale for Depression (Hamilton, 1967) either before or after treatment. Akiskal (1987), commenting on Nandi *et al*'s study, wrote that the "response to tricyclic antidepressants in these 'non-cases' of so-called minor or intermittent depression . . . [suggests] some affinity with morbid depression" (Akiskal, 1987, p. 65). His use of the term 'non-cases' is surprising since Nandi *et al* were clearly searching for major depressions, as indicated by their description of their typical subjects.

Response to treatment of community and clinic cases of depression would have to take into account the possible effects of the 'non-illness' variables mentioned in connection with social incapacitation.

Discussion

There seems to be little evidence that unequivocally demonstrates that community cases of depression differ from clinic cases. Where the data do suggest differences (e.g. the greater number of symptoms and greater social impairment in clinic cases), these could result from the fact that community samples will be diluted because of the variable types and degrees of depression whereas the clinic samples will be of a more selected nature. If this is so, such differences could be expected to be reduced if a higher threshold for caseness in the community samples were adopted. However, this argument would appear to beg the question of the differences between cases of depression in the two settings, and the methodological proposal defeats the whole purpose of the development of instruments such as the DIS and procedures such as the ID. As Wing (1976) noted, the use of the ID to identify community cases according to a specifiable and repeatable procedure makes it possible to investigate questions as to whether theories of aetiology, symptom maintenance and treatment that have been tested on the clinic cases can also be applied to the community cases. Although a number of yardsticks will have to be used in the investigation of the similarities between community and clinic cases of depression, each should be used in the same manner and with the same thresholds in each setting. Whereas threshold points on first-stage screening instruments may have to be changed from sample to sample in order to predict cases on some gold standard (Tarnopolsky *et al*, 1979), thresholds on the gold standard itself should not differ from sample to sample if the nature of the psychiatric cases in the two samples are to be compared or findings from one are to be generalised to the other.

When one uses the same threshold (e.g. an ID of five) to identify cases on a random community sample and a clinic sample, a larger proportion of the former than the latter sample will be below threshold. Wing *et al* (1981) found that 89% of a random community sample were below threshold and Dean *et al* (1983) found that 91% of a random sample of 576 women were below threshold. In both instances the data were obtained with the 40-item PSE. Wing *et al* (1981), using the 140-item PSE, found that 12% of 74 out-patients and 5% of 162 in-patients were below threshold. Sashidharan (1985), also using the 140-item PSE, found that 25% of his clinic cases, which included out-patients and in-patients, were below threshold. Comparisons between the community and clinic samples are difficult because the PSEs used in the two settings are different. It is noteworthy that when data from a 40-item PSE were examined, Sashidharan (1985) found that 45% of the clinic cases were below threshold.

Although the use of the full PSE in the community studies might have resulted in a lower proportion of individuals with below-threshold IDs, and although a considerable proportion of clinic cases are below threshold, there is no doubt that the community samples are more 'diluted' than the clinic cases. However, no matter how much more diluted community samples may be, as a result of the greater proportion of individuals in this sample who will be below case thresholds but who may show varying degrees of distress and symptoms of depression, this does not present a problem for those interested in comparing *cases* in the two settings. Individuals who are below case thresholds may be of interest for some research purposes but are not of interest for those who wish to investigate the similarities and dissimilarities between *cases defined and identified in the same manner* in community and clinic populations. Whether or not the above-threshold community individuals show more variable degrees and types of depression than the clinic patients is an empirical question which has not yet been adequately investigated.

From a public-health point of view, an argument can be made for investigating community cases. Wing *et al* (1981) reported that in a random community sample, those identified as cases had twice as many consultations with their family physicians as the remainder. Brown & Davidson (1978), in a random community sample of 458 women, found that the accident rate per year for children under 16 was 10% for those with mothers who did not have a psychopathological condition, 17% for those whose mothers were cases (mostly depression), and 14% for those whose mothers were borderline cases (ID = 5). Brown and Davidson (1978) noted that the comparable data for a sample of psychiatric patients and a series of women attending family physicians because of depression were almost identical. Brown's (1988) raw data indicated that the accident rate for patients was 16%.

Recognition of community depressions as worthy of investigation does not, however, resolve the problem of the similarity or difference between such depressions and clinic cases. If depressions in both settings are basically the same and one is therefore justified in generalising research findings from community to clinic cases, there are the following advantages to researching the problem in the community: (a) as Eysenck (1970*b*) has noted, the selection processes that bring people into clinics may give rise to unreal associations between symptoms; (b) investigations of patients are often confounded by factors such as medication; and (c) community studies might enable one to study the early development of symptoms.

There are some who suggest that using the same measuring techniques in the community that are used in clinics, and taking a categorical, diagnostic approach to psychopathology in the community may not be fruitful. For instance, a sociologist reviewing Brown & Harris (1978*a*) wrote: "simply to assert the necessary utility of psychiatric diagnostic concepts and procedures in such a way as to exclude from consideration the possibility that they are reflections of institutional practices and ideological divisions, is to imbue them with a facticity that renders them unproblematic. A clear assumption of this study is that if sociologists are to be of use to psychiatrists then they must make use of existing psychiatric knowledge, and must further develop their own knowledge in ways that are acceptable to psychiatrists. While not wishing to decry the desire to be of assistance to psychiatrists, it is being suggested that the underlying methodology and perspective of this study has limited the extent to which a more sociological understanding of a phenomenon such as depression might be developed" (Goldie, 1979, p. 354).

Some psychiatrists have also expressed a similar view. For instance, Williams *et al* (1980) wrote: "at the present time, epidemiologists face the challenge of disengaging themselves from preoccupations with methods of measurement derived from hospital psychiatry, and of facing up to the difficulties involved in conceptualizing and measuring forms of pathology more relevant to the description of the depressed person in the community. If this challenge can be met, it will result in a more realistic and human view of mental functioning than has been achieved so far" (p. 112).

Acknowledging the possibility that the above views concerning the need for new approaches are valid, it may be that a categorical, case-finding (clinical) approach should be supplemented with a dimensional approach in community settings. However, an argument could also be made for increased exploration of the value of dimensional measurements in clinic settings. Since 1950, Eysenck has consistently argued for a dimensional approach to the measurement of psychopathology (e.g. Eysenck, 1970, 1986), and there is some evidence for the usefulness of this. Weissman *et al* (1978) and Hirschfeld *et al* (1986) found that of a large number of variables assessed, only scores on the neuroticism (N) scale of the Maudsley Personality Inventory (MPI: Eysenck & Eysenck, 1964) predicted the course of unipolar depression. High N scores have also been found to predict poor response to behaviour therapy (Lazarus, 1963; Morgenstern *et al*, 1965; Schmidt *et al* 1965; Wolpin & Raines, 1966). However, any data comparing community and clinic cases of depressions in terms of their scores on personality scales (such as the N scale) would not provide an answer to the question concerning the similarities and dissimilarities between the depressions themselves in the two settings.

In general, research designed to compare the nature of depression in the community and the clinic should satisfy the following criteria.

(a) Cases of depression in both settings should be identified using well established, standardised psychiatric interviews and by using the same algorithms for diagnosis.

(b) Interviewers in both settings should have reached the same standard in their interviewing and rating skills. Where possible, the same interviewers should assess both the community and clinic individuals.

(c) The cases of depression identified in both settings should be further classified using classification procedures (e.g. endogenous v. non-endogenous depression, acute v. chronic cases) that have shown promise in investigations of the nature of clinic depressions.

(d) Classes and subclasses of individuals should be as homogeneous as possible. Although, for instance, it is extremely difficult to identify individuals who satisfy criteria for a depressive disorder but not for an anxiety disorder (Costello *et al*, 1988; Stavraki & Vargo, 1986), the nature of depression will not be understood unless we find procedural and statistical strategies to determine the similarities and dissimilarities between community and clinic cases of 'pure' depression. Although depressions in both the community and clinic are likely to be complex phenomena which, for example, coexist with other psychiatric conditions, as Banaji & Crowder (1989) noted: "the more complex a phenomenon, the greater the need to study it under controlled conditions, and the less it ought to be studied in its natural complexity" (p. 1192).

(e) Quantitative data concerning the number, severity and duration of symptoms should be obtained as well as categorical data concerning the numbers of individuals who meet criteria for classification as cases. Care should be taken to analyse separately quantitative data for the depressive and the non-depressive symptoms.

(f) In order to obtain comparable data on the number and severity of symptoms, these data should be obtained for the peak of the depressive disorder. Because of the difficulty of conducting prospective studies designed to enable concurrent assessments of the onset of a depressive disorder, these assessments will probably have to be made through thorough retrospective interviews. However, when the depression identified in the community is not in remission, that individual should be followed up at regular intervals in case the peak of the disorder had not been reached at the first interview. Such follow-up assessments of clinic cases are not likely to produce relevant data because most of the cases will be in a treatment programme shortly after contact with the clinic. The best an investigator can probably do is to be sure that the assessments of the clinic cases are done before the treatment programme starts and do sufficient retrospective probing to at least obtain data on the course of the depression up to the point of entry into the clinic.

(g) If measures of non-illness variables such as social performance are obtained, and given that the goal of an investigator is to determine how similar community and clinic cases of depression are, an effort should be made to demonstrate that differences on the non-illness variables are functionally related to the depression variable. Examining the correlations between scores on the depression and non-illness variables would provide some data that at least would suggest such a functional relationship. More convincing data might be obtained by interview probing as to the reasons for an individual's score on the non-illness variables.

The most general principle that should guide research into the similarities and dissimilarities between community and clinic depression is not to lose sight of the fact that it is the nature of the depression itself, unconfounded with other illnesses and unconfounded with non-illness variables, that must be investigated as thoroughly as possible.

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The Functions of Asylum*

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Many of the functions of large psychiatric hospitals were those of asylum. As the structure of services has changed and the role of the large hospital has diminished, the necessity to continue to cover their functions has tended to be forgotten, partly because it has been thought that, even at best, they were purely protective. Such a point of view cannot be sustained. The functions of asylum have always been both refuge and recuperation. 'Community care' will come to deserve the odium now attached to the worst practices of former times if the tradition of asylum practised in the best of the large hospitals is not (with appropriate modification) acknowledged, properly placed in the psychiatric curriculum, and given high priority in service planning.

When Mr Enoch Powell, then Minister of Health, made his famous speech to the National Association of Mental Health in 1961 he made the following central point:

> "Building hospitals is not like building pyramids, the erection" of memorials to endure to a remote posterity. We have to get into our heads that a hospital is like a shell, a framework to contain certain processes, and when the processes are superseded, the shell must, most probably, be scrapped and the framework dismantled." (Powell, 1961)

This serves as text for a commentary on community care in the 1990s, but for 'framework and process' I will substitute 'structure and function'.

The functions that were being superseded were those of bad institutions – authoritarian, custodial and deadening. They were the reason for the reaction against the idea of institutions more generally. However, as Titmuss (1959) had earlier pointed out:

> "No such swing of opinion away from the *good* institution can be discerned: the effective general hospital for the acutely ill, the public school and other socially approved forms of institutional care. But these have been experienced and remembered only by a minority; for most people institutional life has spelt little besides ugliness, cheapness and restricted liberties."

The Oxford English Dictionary (OED) definition of 'asylum' actually conjures up quite a warm and humanitarian image: "inviolable protection"; "a secure place of refuge, shelter or retreat". One of the illustrations given is a quote from 1728: "A port, where his ships might find an azylum". The functions of asylum are those of a haven: to provide a calm and peaceful environment, protection from violence outside, and a base for repair and reprovision. These functions may be carried out in "a benevolent institution . . . for some class of the afflicted, the unfortunate or destitute". The OED gives several examples of such classes, but adds a rider to that of the lunatic asylum, to the effect that the term 'asylum' is sometimes popularly restricted to this one type.

The labels we give to our concepts should be no more than a shorthand for a more extended description that others can check against their own observations. However, in everyday usage some words tend to take on a connotation that owes more to emotion than to reason. 'Asylum' is one of these. It is often popularly restricted in meaning, if not to an 18th-century madhouse, then to a 'total institution' as pictured by Goffman (1961) – Asylum with an upper case 'A'. The 'community', on the other hand, tends to be seen as a cohesive and caring neighbourhood, although there are very few such in industrialised societies. 'Asylum' then becomes a convenient Aunt Sally, while being in the 'community', by a process of tautology, becomes an administrative goal in itself. I propose to adopt the OED definitions, which allow an impartial examination of the extent to which asylum (lower case 'a') functions are being carried out, whatever the nature of the setting.

'Asylum' in the 18th and 19th centuries

When William Tuke, in 1792, set up an establishment for the 'moral treatment' of the insane he did not wish to call it either an asylum or a hospital. "York already possessed an asylum, operating under conditions which made the use of the term a mockery, and the Retreat was not a hospital." William Tuke had a strong distrust of the medical profession and its methods. Daniel Hack Tuke states that 'The Retreat' was suggested by his grandmother, William's daughter-in-law, "to convey the idea of what such an institution should be, namely . . . a quiet haven in which the shattered bark might find the means of reparation or of safety" (Jones, 1972, p. 47). The functions of asylum that Samuel Tuke described in his *Description of the Retreat*, published in 1813, were very similar to those put forward by Pinel in his *Traité Médico-Philosophique sur l'Aliénation Mentale* of 1801 (Pinel, 1806). Both regarded the functions as twofold – refuge and recuperation.

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To John Conolly (1830) the lunatic asylums and madhouses with which he was acquainted were not fit for the care of the insane, whether acutely disturbed or convalescent: "so long as one lunatic associates with another lunatic, supposing the cases to be incurable, so long must the chances of restoration to sanity be very materially diminished." But in view of the squalour, disease and misery endured by the sane in larger towns, what was the alternative? Scull (1989) quotes MacGill, writing in 1810: ". . . the circumstances of the great body of mankind are of such a nature as to render every attempt to recover insane persons in their own houses extremely difficult, and generally hopeless."

Scull argues: "To improve the living conditions of lunatics living in the community would have entailed supplying relatively generous pension or welfare benefits to provide for their support, implying that the living standards of families with an insane member would have been raised above those of the working class generally . . . something approximating a modern social welfare system, while their brethren were subjected to the rigours of a Poor Law based on the principle of less eligibility." This has a familiar ring to it.

Conolly's antipathy to asylums was restricted to their use for curable cases ("two-thirds, or I might say four-fifths of the cases of reception"), and he was particularly concerned about those "who have been accustomed to refined society" (Conolly, 1830). When he introduced the principle of non-restraint to the large asylum at Hanwell, following Gardiner Hill's demonstration at Lincoln, he seemed tacitly to accept that his earlier estimate of prognosis had been optimistic. Conolly's success at Hanwell provided an acceptable alternative both to the madhouses and to the community neglect of the time, and gave a much needed gloss to the public image of the asylum. In this way he contributed to the growth and systematisation of the asylum system during the next 100 years.

I am not here concerned with the merits and demerits of that system, except to point out that the structure within which the functions of asylum are carried out must, as Scull suggested, always be judged within the context of the social conditions of the time. I would add the context of medical and social knowledge to that. There must always be a balance between the protective and the rehabilitative functions. The reason for beginning with this brief historical sketch is to set the scene for the changes that have occurred since the 1920s, interrupted and then accelerated by World War II.

'Asylum' after World War II

During the late 1940s and the 1950s the foundations of the welfare state were laid in legislation that included every aspect of social life – pensions, family allowances, education, unemployment and sickness benefits, a complex of personal social services, a national health service and provisions for the disabled and the destitute. The 'community' that had looked so threatening and brutalising, especially for vulnerable people, during the earlier stages of the industrial revolution, now seemed more welcoming. Parallel changes, which Alexander Walk called "back to moral treatment", were made in the mental hospitals.

All the techniques of rehabilitation and resettlement now accepted as good psychiatric practice were introduced or reintroduced in hospitals like Glenside, Netherne and Warlingham well before the introduction of reserpine and chlorpromazine. The same is true of admission policies like that at Mapperley, where the emphasis on care outside hospital originated before the war when Duncan Macmillan was medical officer of mental health for Nottingham, as well as superintendent of the mental hospital. However, the success of the new medications, the first really effective physical treatments to be introduced, reinforced the optimism of the time and made it inevitable that the structure of the mental health service must change. Perhaps the most obvious reason for this was that the acute symptoms of psychosis often abated within a few weeks of admission, and, if patients wanted to leave hospital, it was their right to do so. What became known, often disparagingly, as "the early discharge policy" leading to the 'revolving door' system of care, was at many hospitals not a policy at all but an acceptance of the inevitable.

Two theory-driven studies of the clinical and social practice at Netherne, Mapperley and Severalls hospitals, carried out during the 1960s, illustrate the variation in standards at the time. One compared the quality of the care provided in the three hospitals for long-stay women under the age of 60 with a diagnosis of schizophrenia, in order to test the assumption that there would be an association with disability. In 1960, the environment at Netherne was socially rich in most respects, particularly by comparison with Severalls. One of the central factors seemed to be length of time doing nothing, which was strongly associated with severity of negative symptoms, and was least prominent at Netherne. As the environment of Severalls Hospital improved in this respect during the subsequent eight years so did the level of disability of many of the residents who had been admitted in 1960 (Wing & Brown, 1970).

The other study was a five-year follow-up of patients with schizophrenia admitted to the same three hospitals in 1956 (Brown *et al*, 1956). Here the hypothesis was that shorter hospital stay and greater contact with community services in Nottingham would be associated with a more favourable clinical course and less family burden. Unlike the in-patient study, however, no significant differences could be demonstrated. This was partly due to refusals by patients or families to accept follow-up visits, partly because there were not enough agencies to undertake long-term domestic and industrial rehabilitation, and partly because the most severely disabled people did not necessarily receive the most community contact. Such problems were not solved in any of the three areas. In fact, the average amount of time spent doing nothing by patients who were unemployed was higher than that of long-term in-patients in the first study.

A feature of both studies was that the general trends in the results could, with hindsight, have been predicted from a knowledge of the personalities of the medical superintendents of the three hospitals.

The functions of asylum can be specified in some detail from comparative research into the practice, at that time, of the best hospitals and of the new facilities set up outside to supplement their services. The first function (refuge, shelter, retreat, sanctuary) included protection from: cruelty; exploitation; intolerable stress; competition (e.g. if unable to compete for housing or work on the open market, or unable to use ordinary amenities for recreation); pauperism (insufficiency of food, light, heat, clothing and basic personal possessions); social and intellectual poverty and isolation; and harming self or others, whether by self-neglect or violence. The second function, reparation, included: identification of the causes of social disablement, by skilled diagnosis and psychosocial assessment; treatment, within the limits of contemporary medical knowledge, of the physical and mental disorders responsible for admission; and provision, within the limits of local social attitudes and facilities, of the means of rehabilitation and resettlement. In addition, a place was usually rapidly available at times of emergency, however difficult the problem, and all services were provided free.

The most typical structure within which these functions were being carried out, sometimes very well by the standards of the time but often not well enough, was the mental hospital estate, with its limited apparatus of outreach into the community and links to the medical and welfare departments, now abolished, of local authorities. Early in the history of Sunnyside Royal Hospital, near Montrose, a superintendent who gave thought to the question of how much land was needed, suggested four acres per patient. When I attended its 200th anniversary, Sunnyside looked not much more suitable for people trying to recuperate from severe mental disorders than the factory buildings that form the present 'state of the art' district general hospitals. Nevertheless, when working well, the system did have the substantial advantages listed above, as well as two more that tend to be forgotten. There was a relatively limited and identifiable line of responsibility to a physician superintendent at the top, and there were the appreciable benefits of space, trees and grass.

The disadvantages were mainly those of size, cheapness and overprotection: having wards the size of aircraft hangars; the necessity of conforming to timetabled routines because of low supervisory staff ratios; the restriction of individual choice; and the distance (in some cases) from centres of ordinary social activity. Stigma was also attached to the buildings and perhaps amplified the odium that is always accorded to deviant behaviour, whatever the setting. Scandals were not uncommon, usually in some part of the hospital that had become isolated from the rest and was not under vigilant supervision.

How far these advantages and disadvantages were specific to large institutions was a matter for intense debate then as it is now. Two compilations of papers provide a good account of the arguments (Freeman & Farndale, 1963; Freeman, 1965). Since the functions carried out by mental hospitals had changed so radically, Enoch Powell's point about the need to dismantle the structure seemed apposite. He did not say, however, what structure should be put in its place (Powell, 1989).

Several major problems were foreseen. Many long-stay patients lacked the motivation and the skills to achieve their own resettlement. On the hospital estate, or within reach of it, all the steps and landings on the three stairways of residential, occupational and social rehabilitation (Wing, 1986) *could* be provided. The staff, who were needed to help people mount at their own pace or (just as important) descend without falling precipitously to the bottom, *could* be in easy communication with each other.

This is not to say that they were, only that the structure allowed such functions. If services became geographically fragmented, and responsibility diffused between different statutory, voluntary and commercial organisations, integration into a well planned and interconnected whole could be difficult. Co-ordination between dispersed day and residential units would be particularly problematic. Further problems concerned the extent to which professionals would and could be replaced by informal carers (Abrams, 1978) and how far professionals would wish to continue to serve the most disabled patients if a wider choice of work were on offer (Wooff *et al*, 1986).

In order to judge the success of any structure, it is not sufficient only to improve on the standards of an isolated and poverty-stricken 2000-bed Victorian hospital with a chronic staff shortage, poor facilities and a reputation for scandals and neglect. The standard by which to judge any new structure should be fourfold: (a) whether the functions of asylum are being carried out at least as successfully as in the *best* hospital-based services of the past; (b) whether any disadvantages thought to be inherent in the old structure were being avoided; (c) whether problems specific to the new service were being introduced; (d) whether extra advantages were being derived by people who might once have become long-term residents because of an increased interaction with their families and with the general public. Chief of these latter might be enhanced enabling functions that would provide greater choice to handicapped people than could have been provided in hospital and thus lead to a better quality of life.

Very few such studies on new structures have been published. The most comprehensive attempt to provide a full and integrated structure that would allow the catchment hospital to be closed was the Worcester Development Project (WDP). Substantial central resources were put into the area in order to make the demonstration. In particular, new day services were provided in generous measure along with two new acute psychiatric hospital units. The proceedings of the conference called to mark the final closure of Powick are now published (Hall & Brockington, 1990) – the financial, administrative and clinical statements made there will be examined carefully.

There were critical comments at the conference about health authorities that were proposing to close hospitals even though no alternative structure based on the WDP model had been established. However, very few districts in fact have such a range of services. Even if there were, Worcester and Kidderminster are substantially more socially advantaged than the average across the country, and it would be unwise to generalise from them to average or below-average districts. That not all the problems had been solved even in the WDP area was indicated by a story on the front page of the *Malvern Courier*, published during the conference. It began: "Care agencies in Malvern have expressed fears about the lack of support for people who have been discharged into the community from mental institutions." Scandals featured in the press now occur in the 'community' rather than in the hospitals. Torrey (1988) has described experiences of people discharged from American hospitals to an uncaring and hostile 'community' that are as harrowing as any described two centuries ago. Scandals may not be typical of the general level of care but that was also true of the hospital cases featured in the 1960s. Torrey is complimentary about the safety net provided by our own health and social services, but we have no reason for complacency.

Who needs asylum?

Most of the patients who used to accumulate in the large psychiatric hospitals even though their need for long-term high-dependency care had ceased, have now left. Very few new admissions lead to such long-term care. Many of the functions of asylum can be served by a variety of geographically and administratively separated agencies, as long as all the units are in place and there is co-ordination and co-operation to ensure smooth movement and sharing of functions between them. On the other hand, a fragmentation of functions and lack of a strong overall management policy, particularly when added to a scarcity of resources, must lead to disasters. The Commons' Social Services Committee report (1985) and Griffiths' proposals (1988) were designed to prevent this, but it is not clear how the procedures recommended in the White Paper on community care will promote continuity and provide the tough leadership, on a defined geographical basis, that is required.

Whether the functions of asylum can be completely fulfilled without an 'Asylum' as one component of a district service is as hotly debated now as it was 25 years ago. The word itself has been devalued but there is a measure of agreement that, at the bottom of the three stairways that should form the main framework of a comprehensive district structure for people with high-dependency needs associated with long-term mental disabilities, there should be a strong foundation on which the rest of the edifice rests; a haven of needed refuge but also a harbour from which to set out again. The name 'community', in the sense of a small group of people sharing common aims and needs, is much more appropriate to a place that continues the tradition of Tuke's Retreat than to the modern localities that are usually given the name (Abrams & McCulloch, 1976).

Two groups of people, with relatively well defined dependency needs, those with severe mental handicap or dementia, will not be considered here. A third, more heterogeneous group is represented by people with a variety of mental disorders who still, in spite of everything that is done (deliberately or by default) to prevent it, accumulate as long-stay hospital patients. There are many like them who do not acquire this status, but are to be found at home with their families, in bedsits or lodgings, in National Health Service (NHS) or local authority or charitable hostels or homes, in Salvation Army shelters, or in prison on remand or sleeping rough on the streets. It seems much more difficult, therefore, to estimate numbers or to assess their current status than it was in the 1960s. A much larger part of the prevalence is invisible. As the large hospitals run down further, and the small ones focus only on acute episodes, this element of uncertainty will seem to grow.

However, because there has been no major breakthrough in treatment or in methods of rehabilitation since Robertson (1981) made some projections based on trends in hospital statistics up to 1981, his figures do still provide a reasonable estimate of the numbers of people on behalf of whom many (in some cases all) of the functions of asylum still need to be exercised. Because of differing levels of social deprivation across health and local authority districts, no single formula can be applied, but the average for England works out at about 50 per 100 000 population. The kinds of psychiatric and social problems experienced by the people concerned are discussed elsewhere (Wing, 1986; Wing & Furlong, 1986).

So vociferous has been the condemnation of Asylums, and so universal the identification of Enoch Powell's out-of-date structure with the functions that it served, that very little attention, and virtually no experiment, has been devoted to the alternative structures that might serve the functions of asylum. It is a classic case of the baby and the bath water.

Such new structures might take two forms, depending on the availability of sites. Where a large hospital estate is conveniently situated in relation to the population it serves, a section could be reserved for a sheltered community, organised on the core and cluster model, with close connections to other parts of the service. The rest of the estate should be used imaginatively; with sectors devoted to housing, shops and leisure and business facilities that would become part of the neighbourhood as well as being available to patients. Most of the former hospital functions could be served from buildings that merged into the new complex without being specially identified. In this way, the 'community' would be brought on to a site where a mental hospital had stood for a century or more and where local people were familiar with its presence. John Burrell has worked out the architectural basis of the idea in substantial detail (Burrell, 1986).

Another possibility is the free-standing core and cluster model, although this might be at greater risk of becoming isolated from the rest of the district services and from the locality as well. One health region (South East Regional Health Authority, SETRHA, 1988) has adopted the name 'haven' but equated it with the more limited hospital-hostel concept (Hyde *et al*, 1987). At the Maudsley this is developing into a core and cluster arrangement with the central house and most day care on the hospital site and other houses off it (Wykes, 1982; Garety *et al*, 1988). Comparative research has not been undertaken into the merits and demerits of more extensive contemporary forms of sheltered community. But whatever structure is found to be most effective, a comprehensive and integrated district service requires co-ordinated planning, management and finance and a clear-cut policy for caring for the most persistently disabled people. It is essential to understand that such havens must be an integral part of such a district service. If the rest of the system is not in place and working, the name is inappropriate and should not be used (Wing, 1990).

Conclusion

Parry-Jones (1988) is generally sympathetic to the aims and practice of asylum with a small 'a', and sensitive to the swing of the pendulum during the 19th century from neglect to reform to neglect again – and then back during this century as far as reform. On the basis of his analysis, he could have added that the pendulum could still be swinging towards further neglect. If so, a way to stop it is to insist that the functions of asylum be fully incorporated into all the new structures, and tested for their efficacy, as we dismantle the old ones. Although these functions have been elaborated and systematised, they remain much the same as in the time of Tuke and Pinel. We have more effective medical treatments at our disposal which make the task of rehabilitation and settlement easier, and there is beginning to be a respectable body of knowledge about how to help and sustain families. Our social security system, whatever its faults, is immeasurably better than in the time of the Poor Law. However, the needs of severely and chronically disabled people still have a low priority and there is little sign that 'the district' has any therapeutic function in itself.

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Affective Disorders Among Women in the General Population and Among Those Referred to Psychiatrists

Clinical Features and Demographic Correlates

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In a study comparing depressive disorders detected in a field survey ($n = 90$) with patients referred to a specialist treatment setting ($n = 63$), the clinical features and demographic correlates of 'cases' of affective disorders proved to be similar. However, those in treatment settings appeared to have more people achieving definite case status. Hospital-referred cases were also more likely than community cases to be older and single, and this difference persisted even after controlling for chronicity of symptoms.

One of the vexed questions in current research into the epidemiology of affective disorders is the comparability of findings obtained in hospital settings with those based upon general population surveys. The nature and correlates of the depressive disorders of patients are not always consistent with observations on 'cases' found in a general population, and such differences have led to disagreements on underlying theoretical issues. For example, the number, type, and severity of symptoms found in depression, the course and outcome of such disorders, and the presence or absence of putative risk factors are all reported to vary according to the setting from which cases are identified (Paykel *et al*, 1970; Brown *et al*, 1977; Wing *et al*, 1978, 1981; Finlay-Jones, 1980; Bebbington *et al*, 1981; Sashidharan *et al*, 1985). The aetiological and nosological significance of such a diversity of findings has therefore attracted much attention (Bebbington, 1984; Katschnig, 1984).

The importance of comparing and integrating findings from field surveys with those based on patients referred to specialist services cannot be overemphasised. This is especially so in the study of affective disorders, because only a small proportion of those with the disorder are ever seen in hospital. The generalisability of such findings can only be established by comparative studies that include cases from both treatment settings and general population samples. However, such comparative studies are uncommon and, where they are attempted, it is often difficult to draw firm conclusions because of the application of different sets of diagnostic criteria. Even if the variability due to diagnostic differences is reduced, there are two other potential sources of bias. Firstly, there is often scant attention paid to the sampling frames from within which cases are chosen. Hospital cases are generally drawn from new referrals (and hence more likely to be inception cases) while community surveys usually yield cases from cross-sectional samples, and the chronicity of the cases so identified can thus magnify differences. Secondly, various selection factors operate in determining who is referred to hospital (Goldberg & Huxley, 1980). For example, a spurious association could be obtained between a putative risk factor and a disease because of the different probabilities of admission or referral to treatment (Lilienfeld & Lilienfeld, 1976).

This paper examines some of these issues in the context of a direct comparison of representative samples of uniformly diagnosed cases of affective disorder as found in hospital and in the community. The samples are drawn from an epidemiological study that attempted to specify the total morbidity from affective disorder among women (both treated and untreated) in a geographically delimited area of north Edinburgh. More specifically, the aims of this study are stated as follows:

(a) to compare the frequency of clinical symptoms in 'cases' in the two groups
(b) to contrast the distribution of diagnostic categories of affective disorder
(c) to compare selected demographic correlates.

Method

A general population survey of a random sample of 576 women aged 18–65, drawn from the electoral register and with an address in any one of the 12 electoral divisions in the north-east of Edinburgh, was carried out in 1981. Follow-up interviews of selected subsamples of the original cohort were carried out at six months and one year. Details of the design and the methods used in the population study are given elsewhere (Surtees *et al*, 1983, 1986). Case identification and definition were by the Psychiatric Assessment Schedule (PAS; Dean *et al*, 1983), which was based on the first 40 items of the Present State Examination (PSE; Wing *et al*, 1974) and items from the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978). Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978) were applied to all those with symptoms. Cases were identified if they were 'in episode' during the first sweep of the community survey.

All new referrals of women aged 18–65 years to the psychiatric services over six months, commencing 15 January 1981, in the city were identified using the Lothian Psychiatric Case Register (LPCR). All those with an initial diagnosis of affective disorder were eligible. Women were excluded if they had either an address outside the study area, or a primary ICD–9 diagnosis (World Health Organization, 1978) of organic brain disorder, substance abuse, personality disorder, mental subnormality, schizophrenia, or other non-affective psychosis. Samples were drawn from both out-patients and in-patients on the basis of initial clinical diagnosis, availability for interviews, and administrative convenience. New referrals were defined as those who had no previous contact with the LPCR or whose contact (or episode of psychiatric care) was terminated at least six months before the index referral. Out-patient clinics were screened on a rota basis and in-patients were identified using hospital case records. Individuals with a project definition of affective disorder were chosen so that the diagnostic categories used in selecting patients from the hospital sample were the same as those employed in the population survey, although in the hospital study the full interview schedules (both the SADS and the PSE) were used, not the abbreviated versions used in the PAS.

The assessment of community cases was undertaken by trained interviewers, whose ratings of each case were verified and checked by three staff members using audiotapes of the interviews. All the hospital cases were interviewed by SPS. The inter-rater reliability of this rater with the two other staff members (who along with him were involved in verification of the community interviews) on the case/non-case criteria, expressed as an index of the agreement on the presence or absence of a diagnosable psychiatric condition, was 0.77 (Cohen, 1960).

Duration and onset of episodes were assessed in both groups using a set of semistructured questions. Emphasis was given to the start of key symptoms such as persistent dysphoric mood, the time when the respondent was last like her 'normal self' for two months or more, and when the subject first became aware of a change from her 'normal self' and impairment of functioning. For cases from the general population, additional information obtained during their follow-up was also used to date the start and finish of episodes. In contrast, hospital cases had only one psychiatric assessment, but additional information on the start of the index episode was available in some instances from their hospital notes.

Results

The community survey identified 79 women who fulfilled the RDC for a diagnosis of probable or definite affective disorder, that is, major or minor depressive disorder, generalised anxiety disorder, panic or phobic disorder. Forty-nine of this group also achieved an ID (index of definition) of 5 or above (Wing & Sturt, 1978), but 50 women were accorded 'case' status on the basis of symptoms present during the month before the survey, one subject not fulfilling the RDC.

An additional 11 women were identified subsequently as being 'in episode' and fulfilling the RDC at the time of the prevalence survey on the basis of information obtained during the follow-up interviews. Thus, there were 90 women who fulfilled the RDC for a diagnosis of affective disorder in the community.

A total of 351 women from the study area were referred to specialist services and were eligible for sampling (272 out-patients and 79 in-patients), of whom 89 were interviewed using both the SADS and the PSE. On the basis of their psychiatric assessment, 63 women (35 in-patients, 28 out-patients) were found to have a project diagnosis of affective disorder, which included major or minor depressive disorder, schizoaffective disorder (primarily depressive), generalised anxiety disorder, and panic disorder. They all fulfilled the RDC for at least one of these disorders. The 26 who did fulfil the project definition of affective disorder had the following diagnoses: schizophrenia (6), manic/hypomanic disorder (8), unspecified functional psychosis (1), alcohol/drug disorder (5), other psychiatric disorder (4), and currently not mentally ill (2).

The hospital sample constituted 41% of 153 women from the study area who had a clinical diagnosis of affective disorder referred to hospital. In order to establish whether these 63 patients were representative of the 153 eligible for selection, several comparisons were carried out by using the LPCR. There were no significant differences between the two groups in age, marital status, social class, source of referral, history of previous care, usual living group (i.e. number of people living at home), and injury or poisoning preceding psychiatric referral. The only significant difference was in the out-patient group – only one woman had been seen as an emergency, while 24% of all out-patient referrals were emergency or out-of-hours referrals.

Symptoms and syndromes

Using ratings from the abbreviated (40-item) PSE, 50 women from the population survey achieved definite/threshold case status, compared with 46 from the hospital sample of 62 women (one did not complete the PSE) (Table I). While 82% of the community cases are at the threshold, 54% of the hospital cases are definite cases. Mean PSE score (40-item version) for the two groups at ID levels 5 and 6 are broadly similar, although hospital cases at the threshold level have, on average, more symptoms than community ID-5 cases.

Table II shows the distribution of CATEGO syndromes in the two samples according to CATEGO class. The majority of cases from both samples were designated as CATEGO class R or N ('retarded' or 'neurotic' depressions) on the basis of the short form of the PSE. In general the hospital sample shows proportionally more individuals with each CATEGO syndrome, again indicative of the increased number and severity of symptoms in this group. In the hospital group of 'depressives' (CATEGO class R and N) the most

TABLE I
Mean total PSE (40-item) scores in the hospital and community 'cases'

	% at each level		PSE mean scores	
ID level	*hospital (n = 46)*	*community (n = 50)*	*hospital (n = 46)*	*community (n = 50)*
5	46	82	15.3	11.9
6	41	18	22.8	23.3
7	13	–	22.8	–

TABLE II
CATEGO syndromes present in hospital and community 'cases'[1]

	CATEGO class R or N		CATEGO class A	
CATEGO syndromes	*hospital: % (n = 39)*	*community: % (n = 35)*	*hospital: % (n = 7)*	*community: % (n = 15)*
SD (depressed mood)	100	100	29	40
GA (free-floating anxiety)	51	34	100	87
SA (situational anxiety)	26	51	71	87
ED (pathological guilt, etc.)	59	66	29	7
IR (ideas of reference)	36	31	57	13
TE (muscular tension)	87	80	71	67
LE (subjective anergia)	64	40	43	40
WO (worrying, etc.)	100	89	100	80
IT (irritability)	54	71	57	60
SU (social unease)	87	74	86	73
IC (loss of interest and concentration	95	57	43	13
HY (hypochondriasis)	31	3	14	20
OD (somatic depressive symptoms)	82	77	86	53

1. Based on shortened version of the PSE.

frequent syndromes apart from depressed mood appear to be worrying, loss of interest and concentration, muscular tension, social unease, and somatic depressive symptoms. In the community sample also these syndromes predominate (except for loss of interest and concentration). The most striking difference between the two samples is in hypochondriasis, reported by nearly a third of hospital depressives in contrast to only 3% of the community group.

A more detailed comparison of the two groups on the frequency of depressive symptoms comprising the 'endogenous' subcategory of RDC diagnosis of major depressive disorder was attempted next. Of the 79 women in the community who fulfilled the RDC for a definite/probable disorder (on the basis of one-month PAS ratings) at the time of the prevalence survey, 24 fulfilled the RDC for major depressive disorder. The hospital sample yielded 43 women with an RDC diagnosis of major depressive disorder out of 63 subjects.

The RDC category of endogenous major depressive disorder is applied to all subjects who show a particular symptom profile: two groups of symptoms (A and B), a total of at least four symptoms should be present for a *probable* and six for a *definite* diagnosis, including at least one symptom from group A. The four group A symptoms are distinct quality to depressed mood, lack of reactivity to environmental changes, diurnal mood variation, and pervasive loss of interest or pleasure. Group B symptoms consist of feelings of self-reproach or guilt, early morning awakening or middle insomnia, psychomotor retardation or agitation, poor appetite, weight loss, and loss of interest or pleasure in usual activities or decreased sexual drive. Since the PAS does not include items relating to the first two group A symptoms, comparison is mainly on the basis of group B symptoms (Table III). Of the remainder of group A symptoms, pervasive loss of interest was taken as equivalent to loss of interest or pleasure in group B, which was based on item 22, 'loss of interest', in the PSE. The symptom of diurnal mood variation in group A (worsening of depression in the morning, PSE item 27, rated as 2) was present in 14% of the hospital group and 13% of the community sample.

Table III is a detailed breakdown of the frequency with which group B symptoms are present in the two samples. On the whole, similar proportions of hospital and community depressives report the presence of individual symptoms, except for weight loss and loss of interest or pleasure, which are markedly more frequent in the hospital group. Table III also shows that 64% of the hospital group had at least three of the group B symptoms, while only 46% of the community group had a similar profile.

TABLE III

Frequency of six RDC 'endogenous depressive disorder' symptoms in hospital and community cases of major depressive disorder

RDC symptom[1]	*Hospital sample*[2]*: % (n = 42)*	*Community sample: % (n = 24)*
Feeling of self reproach or excessive or inappropriate guilt	57	58
Early morning awakening	43	38
Psychomotor retardation	55	58
Poor appetite	57	46
Weight loss	43	29
Loss of interest or pleasure in usual activities	62	33
All six symptoms present	10	4
Only five symptoms	7	8
Only four symptoms	26	21
Only three symptoms	21	13
Only two symptoms	17	29
Only one symptom	5	8

1. Based on PSE symptoms, except poor appetite.
2. One individual did not complete the PSE.

Diagnosis

The 90 women from the community sample found to be 'in episode' at the time of the first interview are compared with the hospital sample of 63 women in Table IV. Although depressive disorder is the most frequent diagnosis (97% in the hospital group, including schizoaffective category, and 71% in the community sample), the more severe forms (i.e. major v. minor and definite v. probable) occur more often in the hospital group. Apart from the three women with schizoaffective (depressed) disorder, only one other individual had psychotic symptoms (delusions or hallucinations) in the hospital group. In the 43 women with a diagnosis of major depressive disorder in the hospital group, 20 fulfilled the criteria for endogenous depression (12 probable and 8 definite), and 14 had an RDC subdiagnosis of incapacitating depression, 8 agitated depression, and 7 retarded depression (these categories are not mutually exclusive). Because of the limited number of symptoms that were inquired about in the community survey such differentiation into sub-categories was not possible.

Demographic factors

The two groups of women were compared on the basis of their social class, age, and marital and employment status

TABLE IV

RDC diagnosis of hospital and community samples

	Hospital sample		*Community sample*	
RDC diagnosis	n	%	n	%
Schizoaffective depression	3	5	–	–
Major depressive disorder				
definite	32	51	31	34
probable	11	18	22	24
Minor depressive disorder				
definite	3	5	11	13
probable	–	–	1	
Panic disorder				
definite	5	10	2	4
probable	1		2	
Generalised anxiety disorder	8	13	21	23

Note that RDC diagnoses are heirarchical.

(Table V). Married women are those currently living with their husbands. Social class was based on husband's occupation (if the woman was married); if single, her own occupation or that of her father's was used (if living with her father) (Surtees *et al*, 1983). Social class was dichotomised as middle or working class, using a classification according to Goldthorpe & Hope (1974).

In the community survey, out of the 576 women interviewed, 330 (57%) were middle class and 61% were married and living with their husband. Nearly half of the sample (40%) were aged 18–34 years while the oldest age group (55–65 years) accounted for 17%. Over 70% of the women working were full-time or part-time or were students (Surtees *et al*, 1983).

In our comparison of 'cases' found in the general population survey and referred cases, community cases were significantly younger, and there were proportionately more single women in the hospital group.

We also examined the extent to which 'chronicity' of symptoms could have biased the association with demographic factors. Chronicity in this context does not refer to the duration of symptoms alone, but is based on the length of episodes as determined by data available at the time of the interview. For this purpose we categorised the cases identified in each setting as either 'acute' or 'chronic' on the basis of the duration of the current episode, with 26 weeks as the cut-off point. There were 39 women with 'acute' episodes in the hospital sample (62%) and 35 in the community group (39%). Comparisons between hospital and community samples were repeated separately for 'acute' and 'chronic' subjects, and it was found that the differences in age and marital status remained significant.

The design of our study and the methods of case assessment that we employed did not allow us to control for the effect of severity of symptoms on selection of hospital cases. Aggregate symptom score may not be a good indicator of severity of cases here because the community cases are determined according to the 40-item PSE and the hospital-referred cases are designated using the full version of the PSE. For example, it is possible that the presence of a delusion/hallucination with few other symptoms would give a lower aggregate score than a more 'neurotic' presentation, and this would be in contrast to clinical notions about severity of illnesses.

Discussion

Women with depressive disorder as identified in a community survey were not markedly different in terms of their symptoms from those found in a specialist treatment setting. The age and marital status of cases in the two settings were different. On the basis of an earlier study that compared both prevalence and inception rates of affective disorder between the hospital and community settings, we found that older and single women were more likely to be represented in a hospital setting (Sashidharan *et al*, 1988). In this report we show that this demographic difference persists even after controlling for diagnosis.

On the basis of symptom ratings from the 40-item PSE, the CATEGO syndrome profiles of the two samples appear to be similar. The frequency with which particular symptom clusters occur shows only minor variation in the proportions with CATEGO classes R and N; syndromes which were more frequent in the hospital sample were hypochondriasis, loss of interest and concentration, subjective anergia, and free-floating anxiety. In an earlier study (Wing, 1976) where hospital patients with depression were compared with community cases on the basis of all (140) PSE ratings, it was found that there was a paucity of severe and psychotic features in the latter group. Our analysis was based on a shortened version of the PSE, but in contrast to Wing's findings, pathological guilt was present in the majority of community cases and

TABLE V
Proportions of women according to demographic factors

Demographic factor	*Hospital: %* *(n = 63)*	*Community: %* *(n = 90)*	χ^2
Social class			
middle	51	40	
working	49	60	1.3 (1 d.f.)
Age: years			
18–34	30	49	
35–54	49	37	
55–65	21	14	*7.16 (2 d.f.)
Marital status			
single	27	12	
married	44	53	
others	29	34	*8.30 (2 d.f.)
Employment status			
employed	56	59	
unemployed	45	41	0.33 (1 d.f.)

*$P<0.05$.

subjective anergia was experienced by 40% of them. Finlay-Jones (1980) suggested that dysthymic states found in community samples were principally depressed while in declared cases there were more mixed states (i.e. a combination of anxiety and depression). An examination of our CATEGO syndrome profiles however does not sustain this notion. The majority of both hospital and community samples with depression also had anxiety symptoms. We have already reported the absence of 'pure' depressive states in community samples when hierarchical diagnostic rules were suspended or when longitudinal data were examined (Sashidharan *et al*, 1985; Surtees *et al*, 1986).

We have also reported on symptom data that are required by the RDC for a diagnosis of the endogenous subtype of major depressive disorder. Although once again the data available were limited (group A symptoms were not specifically inquired about) the frequency with which six key symptoms occur in the two samples appear to show some similarity. If the co-occurrence of four or more of the group B symptoms is taken as indicative of a diagnosis of endogenous depression, this would suggest that 33% of the community depressives in contrast to 43% of those referred to hospital would qualify for such a label.

Although symptoms in the two samples can be shown to be similar on the basis of abbreviated ratings after partially controlling for the diagnosis, there can be little doubt that the hospital sample is diagnostically more heterogeneous and has fewer people with panic/anxiety disorders. It is also quite likely that if the full PSE was employed in the community study, such cases as were identified probably would not have shown the same frequency of severe psychotic symptoms as found in the hospital group.

A difficulty encountered in this study concerned sample selection. Firstly, although the hospital sample drawn from all hospital contact over six months was representative in terms of sociodemographic and selected clinical variables, it is clear that those referred as emergencies were under-represented. It can be argued that ease of contact was probably determined by acuteness of symptoms and, as a result, the hospital sample was biased towards those with less severe illnesses. We think this unlikely and, furthermore, of the 63 women in this group the majority (35) were in-patients at the time of the study.

Secondly, the community group was a point-prevalence sample, while the hospital patients were drawn from a six-month inception sample. Thus the selection of cases in the latter group was biased towards having more new cases and in fact our data show that the hospital sample reported episodes which were generally of shorter duration than the community group. It is difficult to say how this could have influenced the symptom ratings reported here but, if anything, the community cases would be expected to show fewer acute symptoms when compared with the referred cases. Our data, however, show a similar distribution of symptoms in the two samples.

As far as the demographic correlates were concerned, the differences between the two groups persisted even after controlling for duration of their episodes. Since the differences in age and marital status between the two samples could not be accounted for by symptoms or diagnosis or chronicity of the episodes, it is possible that these could be factors which selectively determine the ease with which individuals progress along the pathway to psychiatric care (Goldberg & Huxley, 1980). A study by Hurry *et al* (1980) which examined the role played by social class in selection into treatment however did not find it had any significant effect on service use.

It would appear that hospital-derived cases of affective disorder and those identified in the general population using similar case-finding methods are not dissimilar in their constituent symptoms. It could be argued that cases from the general population are less severe (most of them at the threshold level of case status) and are diagnostically less diverse than hospital cases, but the similarity between them at the level of syndromes and symptoms is impressive. We offer no evidence to suggest that 'cases' in the community are diagnostically (and by implication in their aetiology or natural history) different from hospital-treated depressives.

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Gender-Mediated Clinical Features of Depressive Illness

The Importance of Temperamental Differences

G. PERUGI, L. MUSETTI, E. SIMONINI, F. PIAGENTINI, G. B. CASSANO and H. S. AKISKAL

In a consecutive clinical series of 538 subjects with primary mood disorders the male : female differences were most skewed (1 : 4) in recurrent unipolars, 1 : 2 in single episode and bipolar I subtypes, and about even (1 : 1) in bipolar II. The sexes did not differ in age at onset of depression, stressors preceding index episodes, endogenous features, psychotic symptoms, suicide attempts, and rates of chronicity. Females had lower mean number of hypomanic, and higher mean number of depressive, episodes. Females also exhibited more anxiety and somatisation, and were more likely to endorse psychopathological items on self-report instruments, which were not reflected in objective measures. Finally, they were more likely to have been admitted to hospital. These gender differences could in part be explained by the higher prevalence of the depressive temperament in women, and of the hyperthymic temperament in men.

Clinical and epidemiological studies have shown a preponderance of females in cases of depression (Weissmann & Klerman, 1977), which is most prominent in non-bipolar samples (Boyd & Weissman, 1981). As women are believed more likely to report depressive symptoms (Silverman, 1968), some authorities have questioned whether the gender differences are relevant to primary mood disorders (Roberts *et al*, 1987).

Data on gender differences in large clinical samples of patients with mood disorders are scarce. Frank *et al*'s (1988) study of subjects with recurrent depression found such differences in several personality measures, depressive symptoms, and in treatment response. Women scored significantly higher on the ego-control scale, while men scored higher on the assertion of autonomy scale; and significantly more men met the criteria for compulsive personality. Furthermore, females showed higher scores in self-evaluation of psychopathology and demonstrated a slower response to treatment. The study did not comment on depressives with a non-recurrent course or those with bipolar substrate, and it failed to consider the temperamental foundations now believed to be relevant to the expression and course of affective illness (Kukopulos *et al*, 1983; Akiskal & Mallya, 1987).

With due attention to these methodological points, the present investigation, undertaken on a very large sample of subjects with primary mood disorders divided on the basis of temperament, polarity and course, addresses possible gender-mediated differences of depression. Such a focus on long-term traits in depression is necessary to explain any gender differences that might exist. Examination of a large sample of depressives referred to a university medical centre can also generate insights on gender-mediated clinical expressions of mood disorders that supplement epidemiological observations.

Method

The sample comprised 538 consecutive patients with clinical depression (167 men and 371 women) seen at the Institute of Clinical Psychiatry, University of Pisa, and at affiliated clinical facilities over a two-year period. The subjects came from a variety of sources, about equally divided between self-referrals, referrals from general practitioners, various medical specialists and psychiatrists, as well as those referred by former patients.

The sample, comprised entirely of subjects with primary mood disorders, was obtained after excluding depressives with pre-existing organic mental disorders, schizophrenic disorders, psychoactive substrate use, anorexia nervosa and somatisation hysteria, as well as those with concomitant panic, social phobic, agoraphobic, and obsessive-compulsive disorders. Diagnostic criteria were based on DSM–III–R (American Psychiatric Association, 1987).

We also excluded those with established neurological diagnoses (e.g. Parkinson's disease, multiple sclerosis, brain tumour), as well as those with serious or disabling medical conditions; however, hypertension under good medical control was not grounds for exclusion. Patients who refused or were unable to undergo standard evaluation, particularly the latter, constituted no more than 2% in this series of studies (Cassano *et al*, 1987). For the assessment of symptoms, psychiatrists completed the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967) and the Retardation Rating Scale (RRS) of Widlocher (1983), and patients completed the Hopkins Symptoms Check-List (HSCL-90) of Derogatis *et al* (1973).

Diagnostic subtyping of subjects was accomplished with the Semistructured Interview for Depression (SID; Cassano *et al*, 1987), which represents a collaborative effort between the Institute of Clinical Psychiatry, University of Pisa, Italy, and the Section of Affective Disorder, University of Tennessee, Memphis, USA. It was developed to diagnose major depressive episodes (MDE; American Psychiatric Association, 1987) and to collect systematic anamnestic data on number and duration of previous episodes, number of suicide attempts, temperamental aspects, residual and interepisodic phenomena, response to previous treatment, 'stressors' related to the onset of the index episode, and the presence of melancholia (or endogenous clinical features), as well as congruent and incongruent psychotic features. Family history data are collected by Winokur's approach as incorporated into the family history method of the Research Diagnostic Criteria (Andreasen *et al*, 1977). The major innovation in this instrument is the provision of operational criteria for depressive and hyperthymic temperaments; these represent the University of Tennessee modification (Akiskal & Mallya, 1987) of Schneider's (1959) descriptions. The depressive temperament requires the presence of at least five of the following items: (a) gloomy, pessimistic, humourless or incapable of fun; (b) quiet, passive and indecisive; (c) sceptical, hypercritical or complaining; (d) brooding and given to worry; (e) conscientious or self-disciplining; (f) self-critical, self-reproaching, or self-derogatory; and (g) preoccupied with inadequacy, failure and negative event to the point of morbid enjoyment of one's failures. The hyperthymic temperament requires at least five of the following items: (a) irritable, cheerful, overoptimistic or exuberant; (b) naïve, overconfident, self-assured, boastful, bombastic or grandiose; (c) full of plans, improvident, rushing off with restless impulse; (d) overtalkative; (e) warm, people-seeking or extroverted; (f) overinvolved and meddlesome; and (g) uninhibited, stimulus-seeking or promiscuous.

Based on exclusion of pre-existing non-affective conditions, family history, subtype of temperament and/or history of hypomanic or manic episodes, as well as recurrence of episodes, the SID decision tree identifies the following subtypes of primary depression modified from the classificatory schema of Akiskal (1983*a*,*b*): (a) classic or bipolar I (BI) disorder with a history of full-blown manic symptoms; (b) bipolar II (BII), defined as MDE preceded or followed by hypomania, or those with pre-existing hyperthymic temperament; (c) bipolar III (BIII), patients not meeting bipolar I or II criteria but who have first-degree relatives with bipolar illness; (d) recurrent unipolar (RU) depression, in the absence of bipolar family history and with neither hypomania nor hyperthymic temperament; and (e) single episode depression. The justification for including major depressives with hyperthymic temperament along with those with definable hypomanic episodes into BII lies in the fact that familial rates for bipolar illness were not statistically different in the two subgroups (8.5% v. 12.3% respectively, $\chi^2 = 0.141$, d.f. = 1, P = NS). The familial rate for bipolar disorders in the two BII groups combined was not statistically different from the rate seen in BI (10.9% v. 17.2%; $\chi^2 = 0.39$, d.f. = 1, P = NS). Thus, SID subtyping permits purer separation of unipolar and bipolar disorders than that achieved in other diagnostic systems.

The SID instrument derives in part from other operationalised procedures with published reliabilities (Andreasen *et al*, 1977; American Psychiatric Association, 1987), as well as new procedures introduced to address the temperamental foundations of affective illness and the assignment of patients into the proposed bipolar and depressive subtypes. We have now systematically used this instrument for over three years in at least 700 interviews, and psychiatrists participating in these series of studies have extensive and systematic training in the diagnostic approach, which has proven reliability (Cassano *et al*, 1987). The semi-structured face-to-face interview, which lasts from 30–60 minutes, is well accepted by patients. Information obtained from patients is routinely supplemented or corroborated by information obtained from significant others who accompany the patient to our clinic; this is of particular relevance for familial psychopathology and the temperamental measures which are based on patient's habitual functioning before the affective episodes. Final diagnostic subtyping is based on all the clinical information obtained in face-to-face interviews by two experienced psychiatrists which is presented to the senior Italian psychiatrist on our team (GBC) who, during a briefer face-to-face interview, assigns consensus diagnoses. The SID variables were compared between male and female depressives. The statistical methods included Student's t-test for dimensional variables, and χ^2 analysis for categorical data. We conservatively used two-tailed statistics.

Results

Our sample included 29 (5.4%) BI, 128 (23.8%) BII, and 230 (42.8%) RU depressives as well as 142 (26.4%) single episode depressives. BIII accounted for only nine patients (1.7%, two males and seven females), and for this reason this group has been excluded from further consideration in the statistical analyses.

As in other depressive populations, there was a preponderance of women in our sample (68.8% v. 31.2%), which was distributed differentially in the four subtypes of depression ($\chi^2 = 18.72$, d.f. = 3, $P < 0.001$): the category of RU depressives had the lowest percentage of males (22.2%), and BII the highest (43.4%). As a result of these differential rates, the male:female ratio was 1:4 in RU depressives, 1:2 in BI and single episode depressive categories, and about even (1:1) in BII (Fig. 1). The even ratio in BII was the same whether one considered BII defined by hypomanic episodes or by hyperthymic temperament

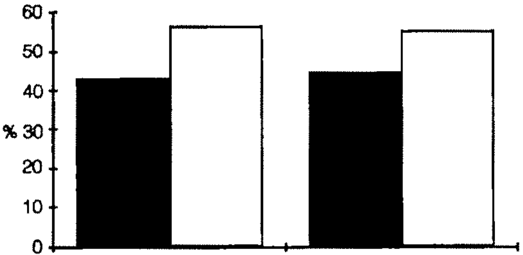

FIG. 1 Number of males (■) and females (□) in SID subtypes of depression.

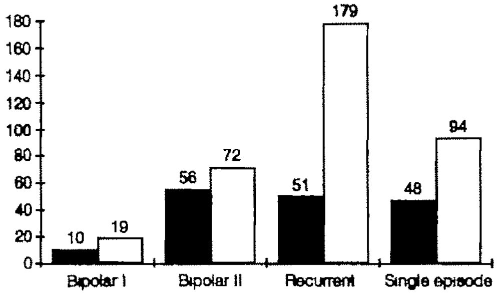

FIG. 2 Percentage of males (■) and females (□) in bipolar II subtypes.

TABLE I
Familial rates of mood disorders, alcoholism, and schizophrenia in first-degree relatives of male and female depressives

	Males (*n = 165*) %	*Females* (*n = 364*) %	χ^2 (*d.f. = 1*)	P
Major depression	34.5	35.4	0.01	NS
Bipolar disorder	6.7	2.7	3.60	0.058 (NS)
Alcoholism	6.1	6.6	0.01	NS
Schizophrenia	1.8	1.1	0.07	NS

TABLE II
Age, age at onset and prior course

	Males (*n = 165*)		*Females* (*n = 364*)		t	P
	X	*s.d.*	*X*	*s.d.*		
Index age	48.9	14.9	51.7	13.4	−2.10	0.036
Age at onset	39.0	15.9	39.8	14.9	−0.55	NS
No. depressive episodes	3.5	3.8	4.4	5.3	−2.19	0.029
No. manic episodes	0.2	0.8	0.1	0.9	0.54	NS
No. admissions	0.7	1.7	1.4	2.9	−3.38	0.001
Age at first admission[1]	37.3	14.9	39.1	13.2	−0.80	NS

1. As not all patients were admitted to hospital, respective *n* values were 59 and 136.

TABLE III
Recovery, history of mania, hypomania, temperament, interepisodic characteristics, and stressors

	Males (*n = 165*) %	*Females* (*n = 364*) %	χ^2 (*d.f. = 1*)	P
Complete remission between episodes[1]	54.8	56.9	0.06	NS
Chronic course (>24 months)	19.4	17.3	0.21	NS
History of mania	6.1	5.2	0.04	NS
History of hypomania	24.2	16.5	3.97	0.046
Hyperthymic temperament	19.4	10.7	6.63	0.010
Depressive temperament	28.5	40.4	6.42	0.011
History of suicide attempts	8.5	14.0	2.72	NS
Stressors[2]	49.1	58.1	3.39	NS

1. The value of *n* is smaller here (male = 104, and female = 260), because, by definition, this item excludes the single episode category and bipolar II patients with only one major depressive episode.
2. Stressors during six months before onset.

(Fig. 2). This preferential clinical expression of male depression in bipolar form is further reflected with a similar trend, albeit non-significant, in their family histories (Table I); familial rates of major depression, alcoholism and schizophrenia were not different in the two sexes.

Regarding age and prior course characteristics (Table II), age of index episode, number of depressive episodes, and number of admissions to hospital were significantly higher in females, whereas age at onset and age at first admission were similar in the two sexes. Table III shows that the two sexes were not different regarding complete remission between episodes, chronicity of depressive episodes, history of mania, stressors during the six months before the onset of the index episode and suicide attempts; by contrast, hypomania and hyperthymic temperament were significantly more frequent in males, and depressive temperament in females. Table IV further shows that, except for two hyperthymic attributes significantly more frequent in males, these temperamental differences in the two sexes were due to the cumulative influence of relatively minor variations in the rates of temperamental attributes which generally favoured depressive attributes in females and hyperthymic attributes in males.

In the 47 major depressives who were classified as BII on the basis of hyperthymic temperament, by definition, 100% were hyperthymic and 0% depressive in temperament. This was not obviously the case with the BII major depressives so defined on the basis of hypomanic episodes, where 21% were hyperthymic and 21% depressive in temperament; and in the major depressives defined as BI on the basis of past mania, 21% were hyperthymic and 38% were depressive in temperament.

Index episodes meeting criteria for melancholia did not differentiate males and females (68% v. 75% respectively); the same was true for psychotic features (8.5% v. 7.7% respectively). Regarding other symptom-related features, females showed higher scores in some but not all objective and subjective measures. Thus, the overall score on psychomotor retardation did not significantly differentiate males from females (17.6 (s.d. 8.7) v. 18.8 (s.d. 8.6) respectively). The total score of HRSD – contributed to largely by the factors 'anxiety-somatisation', 'diurnal variation' and 'sleep disorders' – was significantly higher in

TABLE IV
Temperamental items

	Males (n = *165*) %	*Females* (n = *364*) %	χ^2(*d.f.* = *1*)	P
Hyperthymic temperament				
Irritable, cheerful, overoptimistic or exuberant	40.6	35.7	0.96	NS
Warm, people-seeking or extroverted	57.0	46.2	4.89	0.027
Overtalkative	32.1	37.1	1.02	NS
Vigorous, full of plans, improvident, rushing off with restless impulse	31.5	21.2	6.06	0.014
Naive, overconfident, self-assured, boastful, bombastic, or grandiose	40.6	35.7	0.96	NS
Overinvolved and meddlesome	10.3	6.0	2.42	NS
Uninhibited, stimulus-seeking, or promiscuous	7.9	2.7	0.01	NS
Depressive temperament				
Brooding and given to worry	62.8	70.1	2.40	NS
Conscientious or self-disciplining	76.4	75.8	0.00	NS
Quiet, passive and indecisive	42.7	50.5	2.50	NS
Self-critical, self-reproaching and self-derogatory	37.8	45.3	2.31	NS
Gloomy, pessimistic, humourless or incapable of fun	29.4	37.9	3.17	NS
Preoccupied with inadequacy, failure and negative events to the point of morbid enjoyment of one's failure	28.0	34.1	1.61	NS
Sceptical, hypercritical or complaining	34.1	38.2	0.63	NS

TABLE V
Symptom-related features in the two sexes

	Males (n = *165*)		*Females* (n = *364*)		t	P
	X	*s.d.*	X	*s.d.*		
HAM-D						
Anxiety/somatisation	0.95	0.4	1.05	0.4	−2.79	0.006
Weight	0.48	0.7	0.46	0.7	0.46	NS
Cognitive disorders	0.54	0.3	0.57	0.3	−0.98	NS
Diurnal variation	0.89	0.7	1.02	0.7	−1.97	0.039
Motor retardation	1.76	0.6	1.80	0.6	−0.74	NS
Sleep disorders	0.73	0.6	0.90	0.6	−2.98	0.003
Total HAM-D	19.66	5.8	21.25	5.3	−2.98	0.003
HSCL-90						
Somatisation	1.06	0.7	1.46	0.8	−5.44	0.000
Obsession–compulsion	1.82	0.8	2.03	0.8	−2.58	0.010
Sensitivity	1.08	0.8	1.36	0.8	−3.28	0.001
Depression	1.95	0.8	2.32	0.7	−4.74	0.000
Anxiety	1.42	0.8	1.75	0.8	−4.01	0.000
Hostility-anger	0.79	0.7	0.94	0.7	−1.92	0.056
Phobic anxiety	0.83	0.8	1.15	0.9	−3.99	0.000
Paranoidism	0.93	0.7	1.22	0.9	−3.73	0.000
Psychoticism	0.98	0.6	1.11	0.6	−2.06	0.041

females (Table V); with respect to self-evaluation by means of HSCL-90, females scored significantly higher in essentially all the factors; only for 'hostility-anger' did the higher scores in women represent a non-significant trend.

Discussion

The present study, which focused on a large consecutive series of clinically depressed individuals suffering from different subtypes of mood disorder, confirms the higher prevalence of the depressive syndrome in women reported in the literature (Weissman & Klerman, 1977). Much of this literature, amassed in the field of epidemiology, has focused on gender as a psychosocial mediating variable. Although our sample has the limitation of being focused on cases referred to a university centre, it has the advantage of examining possible mechanisms relevant to the differential expression of clinical depressive pathology in the two sexes.

Before discussing the implications of our findings for such mechanisms, it would be relevant to consider whether these temperamental differences precede or follow depressive episodes. Definitive data on this question are presently unavailable for any type of personality. However, indirect evidence supports the thesis that the temperamental attributes included in the present study develop early in life and represent long-term functioning of the individual (Musetti *et al*, 1989). Studies in both patients and volunteer populations (Akiskal *et al*, 1979; Akiskal, 1984; Depue *et al*, 1981; Eckblad & Chapman, 1986) have shown that these attributes often exist in the absence of major affective episodes. In another 3–4 year prospective study on adolescent as well as prepubertal offspring and siblings of adults with bipolar affective disorder, hyperthymic and depressive temperaments were in evidence before any superimposed episodes occurred (Akiskal *et al*, 1985). Finally, the major depressive sample in the present study included patients with both depressive and hyperthymic temperaments. If temperament represented a post-episodic complication or its evaluation merely reflected state effects of depression, we should not have found any hyperthymic attributes in our major depressive sample. Thus, whatever minor bias the state of depression had on the temperamental diagnoses, it would have been distributed equally between the sexes.

The present investigation attempts to extend previous epidemiological findings concerning depression to include their temperamental foundations. Thus, women had not only higher rates of depressive episodes (and admission to hospital for depression), but also of depressive temperament. By contrast, men had higher rates of hyperthymic temperament and hypomanic episodes. These differences are reflected in the finding of a very skewed 1:4 sex ratio favouring women in recurrent unipolar depressions, and an even sex ratio in bipolar II. This even sex ratio in the BII category is not due to the SID approach which classified all major depressions with hyperthymic temperament into this category; the larger subgroup of bipolar IIs, defined by major depressions with history of hypomania, also had an even sex ratio (Fig. 2).

Manic episodes are known to be more frequent in males (Angst *et al*, 1979); this was also true of our sample, but it did not reach significance because our sample was selected on the basis of index depressive episodes and, therefore, bipolar I patients with predominantly manic expression of their illness were low in number which would, in turn, explain why the overall rate of anyone with a history of mania was relatively low.

Concerning symptom-related features, the presence of melancholia, psychotic features and such core symptoms of depression as psychomotor retardation was not different in the two sexes. However, females showed more anxious symptoms and somatisation as well as an overall higher severity of self-reported symptoms. These findings are open to different interpretation. It is possible that women are more predisposed to develop anxiety and somatisation during major depressive episodes which, in turn, could influence self-report in other symptom-related dimensions. Another possibility is that such gender differences simply reflect socioculturally determined influences on the ways in which men and women perceive their symptoms and respond to them (Briscoe, 1982). Conceivably, men inhibit expression of some depressive symptoms because they are incompatible with the male role or self-image (Warren, 1983). A third possibility is that the higher prevalence of the depressive temperament in females could be responsible for a particular cognitive style with the tendency toward an overestimation of symptoms in self-reported rating scales; by contrast, the hyperthymic temperament favoured by the male gender could explain the tendency to deny or minimise symptoms (Akiskal, 1984; Akhtar, 1988).

The differential distribution of temperaments in our study is consistent with Frank *et al*'s (1988) data on gender differences in personality measures. Although the finding of higher scores on the ego-control scale in females and on the assertion of autonomy scale in males in that study is not completely superimposable on our definition of depressive and hyperthymic temperaments respectively, the findings in the two studies are largely indicative of the same trend in pre-morbid characteristics.

The presence of different temperamental pathologies in the two sexes could also explain, in part, Frank *et al*'s (1988) finding of a more rapid treatment response of depressive symptoms in men and the higher scores in self-evaluation during the remission phase in women. Furthermore, these findings are comparable with the cross-sectional data during index episodes in the present study.

The temperamental differences in the two sexes might represent one of the main mediating variables between gender and the clinical symptoms of depression, their course and prognosis. Indeed, temperamental differences in the two sexes might account for some of the observed gender differences in the rates of depression and its subtypes. Hirschfeld *et al* (1984), who could not demonstrate gender differences in the personalities of recovered depressives, failed to find support for such an interpretation; however, the personality measures in their study did not specifically include the depressive and hyperthymic temperaments as defined in the present study. The population rates for these two temperamental types in males and females are not known. However, data from studies on volunteers recruited through newspaper and classroom advertisement (Depue *et al*, 1981; Eckblad & Chapman, 1986) suggest that temperaments with depressive attributes are more prevalent in females, and those with hypomanic attribute in males. As these attributes develop early in life and are strongly influenced by familial-genetic, as well as by experiential, factors (Akiskal, 1984, 1986), the effects of gender on the subtype and clinical manifestations of depression might have their origin in these temperamental attributes differentiating males and females. More hypothetically, we submit that these temperamental attributes represent the intermediate step between predisposing familial-genetic factors of affective illness and the gender-related clinical expressions of mood disorders (Akiskal, 1986).

The hypothesis that temperamental differences underline the differential gender distributions of major depressive episodes cannot be tested in the present sample chosen primarily on the basis of major depressive episodes; a comparable sample chosen on the basis of mania would be required for that. If verified, this hypothesis would explain Nolen-Hoeksema's (1987) suggestion that gender differences in depression are due to the way in which men and women respond to the fact of having depression, i.e. "men's more active response to their negative moods may be more adaptive on average than women's less active, more ruminative responses".

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The TAPS Project. 4: An Observational Study of the Social Life of Long-Stay Patients

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Observations of long-stay patients in a club in a psychiatric hospital were carried out unobtrusively over several months. Much of the social activity that went on was stimulated by an economy based around the purchase and exchange of tea and cigarettes, and the borrowing and lending of small change. Most patients could be fitted into one of four social categories: helpers, friends, nuisances, and the asocial. The inter-rater reliability of the observations was found to be 0.96. A close correspondence emerged between the observations of patients' social behaviour and material reported by patients when interviewed with the Social Network Schedule.

In July 1983 the North East Thames Regional Health Authority (NETRHA) announced its plan to close Claybury Hospital and partially close Friern Hospital in 1993. Both are large Victorian psychiatric hospitals (Claybury was built in 1893 and Friern in 1851), which had declined from a peak bed occupancy of over 2000 in the 1950s to around 800 in 1983. In 1985, NETRHA set up the Team for the Assessment of Psychiatric Services (TAPS) with the remit of studying and evaluating the closure process. TAPS is supported by funds from NETRHA and the Department of Health, with an initial contribution from the King's Fund. At any one time it comprises about eight full-time research workers from a variety of disciplines.

This paper concerns the long-stay non-psychogeriatric population – individuals with a duration of current in-patient care of one year or more, who, if aged over 65, were not considered to be suffering from dementia. Patients under the age of 65 with dementia or other organic brain conditions were included in the sample, because of their significance for a policy of reprovision of services outside of hospital. The sample consisted of 770 patients, 397 in Claybury and 373 in Friern. The research strategy was to conduct a baseline assessment of the 770 patients in the sample, and then to follow up those discharged to community provisions and compare them with matched patients remaining in hospital.

A batch of seven schedules was selected for the baseline assessment on the grounds of their relevance for the questions being asked. One of these related to the patients' social networks and stemmed partially from the guidelines laid down by NETRHA for reprovision. One of the few stipulations was that patients should be discharged along with their friends. This raised the deceptively simple question of how to define the friends of long-stay patients. Another important question was whether discharged patients would begin to establish social relationships with ordinary members of the public, a change which ought to be reflected in the composition of their social networks. These concerns prompted the search for an existing instrument to describe the social networks of psychiatric patients.

The Social Network Schedule (SNS)

Development of the instrument

The literature about social networks in mental illness contains contributions from many different disciplines. Social anthropologists, sociologists, psychologists, and psychiatrists have looked at ways of evaluating the social contacts and supports of both the mentally ill and the normal population. A review by Beels *et al* (1984) summarises their different approaches, including those of anthropologists like Hammer (Hammer *et al*, 1978; Hammer, 1981), who used an extensive interview with patients, and Sokolovsky *et al* (1978), who used interviews and participant observation to determine the social networks of ex-hospital patients and others in a New York hotel. Tolsdorf (1976) (a psychologist) reviewed the differing variables of structure, content and function in the social networks of healthy and schizophrenic men, while Henderson *et al* (1980) developed the Interview Schedule for Social Interaction, which was intended for neurotic patients. Pattison (a psychiatrist) investigated the networks of neurotic and psychotic patients in the development of a psychosocial kinship model to use in family therapy (Pattison *et al*, 1975).

This and other work has supported the concept of social networks as being potentially protective against stress and repeated breakdown, and has provided indications of core questions about quantity and quality of networks. However, each of these studies used data-collection techniques that were too time consuming for the study of such a large population, and that in many instances were not applicable to long-stay patients. A technique that later came to our attention, multidimensional scaling (Harries *et al*, 1984), was practical and applicable to long-stay patients, but only if they lived with a limited group of people. Consequently, we were impelled to develop a questionnaire of our own that could be used with long-stay patients and took half an hour or less to administer. Earlier versions of the instrument were piloted in separate studies of both hospital and community patients and the current instrument is the seventh draft.

The inquiry begins with a time budget of the previous day and the previous weekend. This aims to elicit the names of any usual social contacts and the settings in which they occur. It emerges from this part of the inquiry whether the patient spends any time off the ward and whether this is occupied with structured (e.g. industrial therapy) or unstructured activity (e.g. the club, shopping). Some patients are unable to provide the names of people they spend a great deal of their time with (e.g. eating together daily) but can identify them by their role (e.g. the man who sits next to me at meals). This is acceptable, and 'named contacts' include those identified by role alone.

Initially, we used the SNS to interview patients in the baseline sample and their care staff, because we anticipated refusals from some patients to answer the questions, although we emphasise the confidentiality of their replies and maintain this strictly. It soon became apparent, though, that there were major discrepancies between staff and patients' accounts. The staff tended to report more significant social relationships on the ward than the patients did, and knew nothing of patients' social activity off the ward. We considered it more important to rely on the patients' perceptions of their relationships on the ward, and since many significant contacts occurred in other locations, we decided to abandon the staff interview with the SNS. This meant that we obtained no SNS data from 281 patients in the sample (36%), but we considered it preferable to accept this deficit than to attempt to fill the gap with data of dubious meaning.

Once the time budget has been completed, the patient is asked for the names of anyone else in each setting he has been in contact with in the past month. The time budget provides a structure which helps patients to recall social contacts. Finally, an inquiry is made about any letters or telephone calls received. This part of the interview delimits the social network of the patient, which contains all the people contacted at least monthly, whose name he can provide, or else whom he can identify by their role. For each person on the list, the patient is asked whether they are a relative, a member of staff, a provider of a service (e.g. shopkeeper) or another patient. Each of these categories has a specific code.

The next step is to ask a series of questions about each social contact until the list is exhausted. Contacts seen less than monthly are not inquired about further. This procedure can be tedious, particularly with lists of 20 or more names. However, most patients soon learn the routine sequence of questions, which can then be dealt with quite quickly. The order of questions is as follows:

(a) How often do you see X? (Daily/weekly/monthly?)
(b) Would you miss X if you never saw him/her again? (Yes/no.)
(c) Would you visit X if either of you moved from here? (Yes/no.)
(d) Do you just say hello to X, do you do things for each other, or do you have conversations? (Passive/intermediate/active.)
(e) Do you consider X to be a friend or just an acquaintance? (Relative/friend/acquaintance.)
(f) Can you talk to X about your personal worries or feelings? (Confidant/non-confidant.)

Question (d) addresses the nature of the social contact between the patient and the named person. We regarded a conversation, however brief, to be more significant than a mere greeting, but where do non-verbal transactions fit into this scheme? In a psychiatric hospital non-verbal transactions, such as lending and borrowing cigarettes, are important social acts. Westermeyer & Pattison (1981) used behavioural transactions, such as begging and receiving food and shelter, to indicate stable or recurring relationships. Sokolovsky & Cohen (1981) also emphasised the importance of exchanges of key goods or services in cementing relationships, but chose to exclude non-verbal interactions from their criteria for network membership. Nevertheless, their division of social interactions into conversational, non-verbal, and salutatory, is identical to ours. We made an empirical judgement that non-verbal transactions were more significant in establishing and maintaining relationships than simple greetings, but less important than conversations. Hence they represent the intermediate category between passive and active interactions in this hierarchical scheme.

Question (f) also merits some discussion. Most patients found little difficulty in deciding whether they could share intimate matters with a particular named contact. However, we were soon faced with making a decision about the sharing of delusions. Some patients took the word 'worries' in the question to mean their psychotic experiences and told the interviewer which of their social contacts they would tell about their delusions or hallucinations. After some discussion we decided that on this basis a legitimate distinction was being made by the patients between confidants and non-confidants.

Our choice of variables (b)–(f) was based on the assumption that they were organised in the form of a hierarchy of social bonding which was pyramidal in shape. Confiding was considered to occupy the apex of the pyramid, representing the strongest and least common bond. Friendship was assumed to occupy the next level, and so on, although we were uncertain about the relative positions of the activity dimension, visiting and missing.

Psychometric properties of the SNS

We have already discussed the problems with using another informant for SNS data. As a consequence, we do not consider interinformant reliability to constitute a test of the schedule itself. Inter-rater reliability has been checked every two weeks with the whole interviewing team throughout the study, with virtually perfect agreement on all items. For example, four TAPS members simultaneously rated SNS interviews with two patients who named 15 and 13 contacts each. Out of 300 and 260 possible disagreements, perfect agreement between all four raters was achieved in 94% and 99% respectively. Another patient with 12 named contacts was rated by five raters, generating 300 possible disagreements, out of which 97% were perfect agreements. The level of disagreement is too low to apply standard tests of inter-rater reliability such as kappa, but for that reason they are redundant. Such close agreement is possible because once the named contacts have been established, the patient's responses are single-word answers which are coded directly. The test–retest reliability of the SNS remains to be studied, and data collection is under way.

Important questions need to be answered about the validity of the information recorded, the structure of the data obtained, and possible redundancy of the items inquired about for each social contact. The issue of validity was tackled by mounting an observational study, reported on in this paper. The other two questions were approached through statistical analyses of the data from the baseline sample, and are the subject of the subsequent paper (this issue, pp. 848–852).

An observational check on the validity of the SNS

The only way to discover whether the information provided by patients on the SNS is valid is to observe them interacting with others in a naturalistic setting. It was, therefore, decided to observe a group of patients unobtrusively and record their social interaction, collecting data on the quality and quantity of their contacts, which could be cross-referenced to data from the SNS.

Assessing a patient's social network in a big hospital by observation presents certain difficulties. It would have been impossible and undesirable to follow patients around all day, so we investigated possible places for observation. In the wards and the workshops there was very little social contact that was not directed at or by members of staff, so we decided to concentrate on places where patients were essentially unsupervised. We discounted the high street and the hospital grounds for practical reasons and chose the larger of the two hospital coffee bars. This is called the club and is run by the Women's Royal Voluntary Service. It is near the front gate, away from the main building, and as well as selling tea, coffee and sandwiches, it has a shop for toiletries, cigarettes and sweets. It is open most of the day and some 20 patients spend the majority of their time there. These patients formed the group whose social networks were to be observed. The overall impression of the place is that of a quiet pub or railway waiting-room.

Initial observations proved that some adjusting to the low level of social activity was necessary, and ten orientation sessions were undertaken. Three problems presented themselves. The first was how to remember who was who from week to week without directly asking the patients their names. It was decided to give each patient a pseudonym based on some impression or physical feature. Sometimes a real name was overheard in a greeting but generally the pseudonyms were easy to create although often forgotten if a patient was not seen for several weeks. In reporting this material the pseudonyms will be replaced by letters.

The second problem was the patients' manifest interest in a complete stranger in their midst, watching and writing. The interest lasted for a couple of weeks and faded, since we gave bland, negative responses to all questions and refused to give in to any begging (new faces are always inundated with begging and often give in as a way of making friends, but they soon learn that this does not work). Often saying 'no' increased the intensity of begging as a 'no' was better than no response: a definite 'no' was simply a shake of the head. After several weeks we were treated as patients ourselves (helped by dressing the part and avoiding eye contact, to maintain a low profile). The observer told CM that he was writing a book and he always inquired how it was going.

The final problem was how to interpret the behaviour being observed. Because of the low level of social interaction, it was difficult to discriminate between continuous meaningful contacts carried on from day to day and chance encounters. In some cases this was easy (for example, when two patients always sat together at the same table). In others, what appeared to be intimate conversations were in fact chance encounters (on occasion patients have sat next to the observer and commenced intimate monologues about other patients). Assessing social contacts by body language is difficult because of the patients' poor movement and posture, due to medication and, it would appear, also to poor consciousness of body posture. So the orientation sessions allowed the observers to develop categories of behaviour, a short-hand method to record data with, and the experience to give observed behaviour its appropriate social weight.

These orientation sessions were written down in long hand, recording as many of the social happenings as possible from every patient present. Here is a typical session example.

'HF and JB are sitting at their usual table. AC and G are sitting together. A male patient sits down and is told to go away by G. He ignores her. AC deserts her. She is not happy about this. SG offers his newspaper to others at the far end of the room. He sits with BB, W and BH. CM is collecting cups. G tells the unwanted guest at her table that he should bathe every day. HS and CS are talking to a female patient. A group of unknown patients are sitting talking with C. G asks me why I am writing notes about the patients. I say I am a student and that it's all part of my training. H is having terrible trouble trying to roll a cigarette on the floor. He gives up and holds his head in his hands. P is doing the rounds saying hello to everyone. She gets complimented on her new shoes. A female patient in a pink dressing gown complains to another about someone on her ward who is driving her mad. Her friend asks her if she is alright and they talk about visitors, divorce and Giro cheques. The first one asks "Will you find another husband?" – "No", the other replies. P and B are buying tea. A female patient comes in, walks straight up to HF and JB and asks them if they want to buy something invisible which she is holding out to them in her hand. Without waiting for a reply she walks straight out again. C gives HS the change from buying a can of orange. HF makes to kiss JB and is told "No, don't". G asks the observer how she can make her stomach grow smaller, since having seen starving children on the television with distended stomachs, she thinks that dieting would only make her stomach larger'.

That account covered everything that happened in about 30 minutes of observation.

It is worth noting that all but the most isolated patients are participating in an economy based around tea, cigarettes and small change. About 80% of patients chain smoke. Lending, giving and receiving are socially functional in the indication of friendship, toleration, sympathy, caring and the desire for contact. A patient can update the state of his relationships by a quick round of begging and its proceeds. Patients appear to have little to talk about, so the constant economic exchange of tea and cigarettes provides much of the substance of contact and communication.

Categories of social behaviour

Analysis of the observations made during the orientation sessions led to the development of four categories or types of social behaviour into which most patients fitted. These are 'helpers', 'friends', 'nuisances', and the 'asocial'.

(a) The helpers collect the dirty cups, empty the ashtrays and rubbish bins, help the asocial patients (with tying shoelaces or buying tea), police the nuisances, and defuse arguments. They circulate or patrol much of the time and seem to be the least disabled.

(b) The friends are patients who have a well defined group of companions with whom they spend their time, sitting chatting and drinking tea, although often with long periods in silence. They do not wander about much but stay in their group, which may have other patients loosely attached to it.

(c) The nuisances spend their time begging for money, cigarettes and tea in a blunt and repetitive manner and tend to be shunned by the other patients. When not begging they sit or wander alone drinking tea dregs or picking up cigarette butts.

(d) The asocial have obvious disabilities and difficulties, for example with buying tea, and are generally ignored or sometimes helped. They sit spaced evenly around the walls, smoking or staring into space in silence. They do not initiate social interactions.

These groups represent a social hierarchy which relates to each patient's quality and quantity of social contacts. It seemed that the helpers had the greatest number of contacts and the asocial the fewest. This hierarchy or organisation was not imposed by staff, and seemed to have evolved to fulfil certain functions in the milieu of the club in a self-regulating way.

Contact rates for helpers might average one in five minutes, for a friend almost continuous contact, for a nuisance one in two minutes, for an asocial one or none in 15–20 minutes. These rates varied, depending on how many patients were present, the time of day, and the weather. Those who wandered made more contacts than those who were sitting, although the sitters had more conversations. So quantity and quality of social contacts were seen to be related in the following way:

(a) the helpers – high quality and quantity of contacts
(b) the friends – high quality and low quantity of contacts
(c) the nuisances – low quality but high quantity of contacts
(d) the asocial – low quality and quantity of contacts.

Using these categories, 12 patients were targeted for observation, two helpers (male and female), two groups of friends (three in each group), two nuisances (male and female), and two asocial.

The ten one-hour observation sessions followed the same pattern as the orientation sessions, except that data were scored on a rating sheet instead of in the form of a longhand narrative. A second observer was inducted into the categories of social interaction to be recorded in order to test the inter-rater reliability of the observations. She made independent observations of the time spent socialising by the 12 targeted patients.

Results

The correlation coefficient between the raw data collected by the two observers was 0.95, while a t-test showed no significant difference between their mean numbers of observations (observer $1=156$, observer $2=153$, $t=0.3$). Hence the inter-rater reliability was extremely satisfactory.

The complexity of a patient's network was calculated by adding up the total number of different patients that were contacted. This gives a score for known people in the patient's network. From the observations was calculated the proportion of each patient's time that was spent in social activity. These data are shown in Tables I and II for the ten patients who also completed the SNS. The network size ranged from 2 to 19, with a mode of 8 and a median of 8. This is closely comparable with the baseline SNS data for the populations of Friern (median = 8) and Claybury (median = 9) Hospitals. M was originally classed as 'asocial', but observation revealed him to be more of a 'nuisance'. He spent a lot of time in the club walking round in circles, arms clasped behind his back, laughing or crying. He was a polite but insistent beggar, and would sit with a benefactor for the duration of the tea or cigarette received, but rarely conversed and quickly went back to pacing up and down. He had no apparent friends or regular benefactors. Two short conversations with an unknown old woman amounted to his closest observed social contact. His network size reflected his begging activities. He shared the combination of a moderately sized network and little time spent socialising with S, a clearly identified nuisance.

Most of the 12 people on whom observations were focused had already been interviewed with the SNS as part of the baseline survey of the long-stay population which took place between 1985 and 1987. It was considered necessary to repeat the interview, however, in case changes had occurred in the patient's networks during the intervening period. It was possible to administer the SNS to 10 of the 12 subjects. One (HS) refused, while W proved to be a healthy employee of the hospital. It needs to be stressed that the SNS covered the totality of the patients' networks, not just their contacts in the club. Hence the

TABLE I
Network size derived from observations in the club and from the SNS

Patient's pseudonym	*Status*	*Network size*	
		Observations	*SNS*
CM	Helper	19	9
S	Nuisance	12	13
P	Helper	11	20
BM	Friend	10	24
BB	Friend	8	16
CS	Friend	8	8
C	Friend	8	4
JB	Nuisance[1]	7	4
M	Asocial	7	2
L	Asocial	2	0

Spearman's rank correlation = 0.74, $P<0.05$.
1. Although originally classed as a nuisance, she developed a friendship during the course of the orientation sessions.

search for any relationship between the SNS data and the direct observations rests on the assumption that social activity in the club is representative of social activity in all other settings.

The SNS generates the number of named contacts seen at least monthly. It was considered that this measure of network size could be directly compared with the network size derived from observations in the club. This comparison is shown in Table I, ranked in order of observed network size.

It is more difficult to decide on a measure from the SNS which would be equivalent to observations of the proportion of time spent socialising. After deliberation we chose the number of friends, on the basis that a person is likely to spend more time socialising with friends than with others. However, we recognised that this is far from ideal, since it is possible to spend a large amount of one's time with one or two friends. The relationship between these two variables is shown in Table II, ranked in order of the proportion of time spent socialising.

TABLE II
Percentage of time spent socialising in the club, and number of friends according to the SNS

Patient's pseudonym	*Observations: % time spent socialising*	*SNS: no. of friends*
BM	75	18
JB	72	1
C	69	3
BB	58	12
CS	53	2
P	33	1
CM	29	6
S	14	11
M	7	0
L	0	0

Spearman's rank correlation = 0.54, NS.

In fact the rank correlation is surprisingly high considering the approximations in the measures, although not statistically significant. The two subjects who stand out as most misplaced when ranked by number of friends are JB and S. As noted above, JB had been asocial, but found a boyfriend with whom she spent the majority of her time in the club. When she was interviewed with the SNS it was clear to the interviewer, a psychiatrist, that she was psychotically depressed, and in fact she was readmitted the following week. S identified every named contact in his network as a friend, apart from his parents. It is likely that he uses this term indiscriminately, and it is not surprising that it fails to reflect his observed social interactions, which largely occurred through his begging activities.

Discussion

Using the SNS, it was possible to obtain social network information from nearly two-thirds of the long-stay non-demented population of the two hospitals. We do not think that the lost third can be ascribed to a fault in the SNS, since in a very similar proportion of patients it was not possible to complete a psychiatric examination using the Present State Examination (PSE; Wing *et al*, 1974). In fact, of the 279 patients with no SNS, 219 also had an incomplete PSE. We have found that inability or reluctance to participate in an interview is indicative of poor social skills (see subsequent paper, this issue, pp. 848–852). Hence the lost third of the population contains a disproportionate number of asocial patients.

The observational study in the patients' club fulfilled two functions. It constituted a method of validating the data obtained with the SNS, and it provided insights into the social life of chronic psychiatric patients living in a large institution. Each of these aspects merits discussion.

Understandably, there is doubt about the ability of long-stay psychiatric patients, the majority of whom suffer from schizophrenia, to report their social relationships accurately. Therefore, it was doubly important to carry out a study of the validity of the SNS. The patients' club was chosen as the location of observations for practical reasons, and because virtually no social interaction between patients was seen to occur on the wards or in the workshops. In itself this is an interesting observation, suggesting that patients feel more free to socialise with each other when no staff are present. An alternative explanation is the absence of an economy of sale and barter in the wards or workshops. The purchase and exchange of commodities, such as tea and cigarettes, appear to serve a social function for this long-stay population equivalent to that of conversation for healthy people. Data from the SNS revealed that non-verbal transactions were utilised by the patients as indicators of friendship to almost as great an extent as conversations. The proportion of individuals designated as friends was 59% of active interactions, 48% of non-verbal transactions, and 23% of passive interactions ($P<0.001$).

We did not expect to find a one-to-one relationship between SNS data and observations of social activity in the club, since the latter represents only a portion of the patients' possible sphere of social interaction. It was surprising, therefore, that such a high correlation was found between the size of patients' social networks derived from the two different sources of data. The correlation between social activity in the club and the number of friends reported on the SNS was lower, and not statistically significant, but should not be dismissed on that account. Firstly, the SNS contains no direct measure of the amount of social activity, and the number of friends can be considered as only a rough approximation to this. Secondly, the correlation found for these long-stay psychiatric patients is even higher than comparable figures derived from the study of healthy people.

Bernard *et al* (1979) compared the actual communication in four naturally occurring groups of between 34 and 58 healthy people with the individuals' own report of their communication. They found that agreement between reports of interaction and actual behaviour was never higher than 50%. They considered that such low agreement cast doubt on the value of reports of social interaction. Romney & Faust (1982) reanalysed the data from the group of 34 people used by Bernard *et al* and calculated the average correlation between verbal behaviour and observed behaviour, which they found to be 0.47 (99% confidence limits 0.41–0.53). They regarded this as indicating that one *can* predict the observed behaviour structure from recall data.

In the case of the patients in our study, we were not only asking them to recall whom they had interacted with in the past month, but also to make judgements about the quality of each social relationship. It was the comparison of these qualitative judgements with actual behaviour that produced a correlation of 0.54. This suggests on the one hand that the patients' judgement was at least as reliable as healthy people's recall of actual social events, and on the other that reliability may be improved by combining qualitative with quantitative data.

The observational study also provided ample evidence of an active social life among long-stay patients, thus endorsing the conclusions of Sokolovsky *et al* (1978) derived from studying ex-patients living in a Manhattan hotel. They noted that many of the residents, "including those with chronic psychotic backgrounds, are involved in autonomously developed social networks and social organization – the extent of which sharply contradicts both the stereotypes of single unattached individuals and schizophrenics as reclusive and extremely isolated". There were undoubtedly some isolated, asocial individuals in the club, comprising up to a third of the attenders. However, there were also small groups of friends, acting as hubs of social activity, and circulating between them and the asocial individuals were the helpers and the nuisances. Both the latter made contacts with the asocial people as well as with the friends, and in this way acted as facilitators, who gave some cohesion to the heterogeneous population of the club. In view of the important role of nuisances revealed by this study, we have included an additional question in the SNS as to whether social contact with an identified individual is welcome, neutral or unwelcome. It is difficult to see how helpers could be identified from the SNS.

The typology derived from the observational study has important implications for reprovision. Firstly, it has become apparent that the social interactions of long-stay patients can change dramatically over short periods of time. JB altered her status as an asocial individual by attaching herself to a boyfriend, HF. The social behaviour of S also underwent a change during the study, when a benefactor in the community gave him money, which reduced his begging and enabled him to begin making friends. These observed changes raise the hope that asocial patients may develop social bonds, given the right atmosphere. But how can we identify its nature and how may we provide it? We believe that the heterogeneity of the population in the club is itself a clue. It seems likely that an admixture of friends, helpers and nuisances is necessary in order to sustain a group of asocial patients, for whom social life may otherwise be completely extinguished. There is evidence that the process of reprovision involves the selective discharge each year of the most socially able patients left in the hospital (NETRHA, 1988). If this continues, it is inevitable that at some stage only the asocial patients will be left. We have serious doubts about their ability to form viable social groups in the community.

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Authors' details will be found at the end of the following paper, p. 852.

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The TAPS Project. 5: The Structure of Social-Network Data Obtained from Long-Stay Patients

J. LEFF, C. O'DRISCOLL, D. DAYSON, W. WILLS and J. ANDERSON

The Social Network Schedule was successfully completed on 489 (64%) of 770 long-stay patients in Friern and Claybury Hospitals and the data on 3774 social contacts of the patients subjected to log-linear modelling. This showed that the category 'confidant' was salient in understanding the data. Latent-class analysis highlighted the importance of the nature of the social interaction between patients and their contacts. In particular, non-verbal exchanges of goods and services emerged as important in creating social bonds.

The development of the Social Network Schedule (SNS) and its content are described in the previous paper (this issue, pp. 842–848). The observational study reported in that paper indicated that the data obtained with the SNS from patients, despite a possible distortion produced by their psychiatric abnormality, are at least as valid as those derived from samples of psychiatrically healthy people (Romney & Faust, 1982). This justifies subjecting the data provided by the patients to statistical investigation of its structure.

Our purpose in conducting the analysis was twofold: to determine whether any of the items in the SNS were redundant and could be deleted; and to see whether members of the patients' social networks formed distinct groupings with differing intensity of social relationships. This would be expected if patients' social relationships resembled those of healthy people.

The data set

Of the 770 patients, it was possible to complete an SNS on 489 (64%). The remainder were uncooperative, mute, or incomprehensible. Because of their difficulties in communicating, it seemed likely that the patients without an SNS have small or non-existent social networks. To investigate this possibility, the patients with a complete SNS were compared with the remainder on two items from the Social Behaviour Schedule (Sturt & Wykes, 1986) which indicate sociability – an ability to initiate conversation, and an ability to respond to a social approach. The information required to rate these items is provided by staff, and is therefore available even for uncooperative patients. For both items severe problems were significantly more common ($P<0.001$) in patients with no SNS than for patients with a complete SNS, thus confirming our assumption that the former are more handicapped socially than the latter. This must be borne in mind when interpreting the data from the successful SNS interviews.

The 489 patients generated a total of 3774 named people with whom they were in contact at least monthly, an average of eight per patient. Complete data were available for 3769 contacts, on which statistical analyses were conducted using the dichotomous variables missed/not missed, friend/non-friend, confidant/non-confidant, and the trichotomous variable for quality of interaction active/intermediate/passive. The variable concerning visiting was not included, as it was discovered that it had been inquired about in different ways during data collection. The instructions have since been amended to clarify this issue.

Log-linear modelling was employed to explore the relationships between the variables in the SNS and to identify any redundancy of items. A number of models were fitted to the data, using χ^2 values generated by each to compare their goodness of fit.

Results of log-linear modelling

The best-fitting model was found to involve three first-order interactions and one second-order interaction. Omitting any one of these interactions resulted in a substantial reduction in the goodness of fit of the model. The various interactions are displayed in Tables I and II.

TABLE I
Log-linear modelling of SNS data: first-order interactions

	No. of contacts		Probability of being a confidant
	Non-confidant	Confidant	
(a) Confidant × missed			
non-missed	1611	126	0.07
missed	1217	815	0.40
(b) Confidant × friend			
non-friend	1710	267	0.14
friend	1118	674	0.38
(c) Confidant × activity			
passive	1013	33	0.03
intermediate	622	101	0.14
active	1193	807	0.40

TABLE II
Log-linear modelling of SNS data: second-order interaction (activity × friend × missed)

Activity	Probability of being missed		Ratio of probabilities of being missed (friend: non-friend)
	friend	non-friend	
Passive	0.56	0.21	2.7
Intermediate	0.63	0.29	2.2
Active	0.80	0.56	1.4

The first-order interactions all involve the confidant variable, suggesting that it is salient in understanding the SNS data: it interacts significantly with each of the other three variables. Contacts who would be missed are nearly six times more likely to be a confidant than those who would not be missed, while friends are nearly three times as likely to be a confidant as non-friends. On the activity variable, the likelihood of being a confidant is virtually nil when social interaction is passive. Indeed, common sense suggests these to be mutually exclusive categories, since confiding is inconceivable if interaction is confined to greetings. Social contacts with active relationships are nearly three times as likely to be confidants as those with intermediate relationships. It is worth emphasising that the probability of being a confidant is almost identical whether the social contact would be missed, is viewed as a friend, or participates in an active relationship.

The second-order interaction involves the three variables other than confidant. It can be seen from the end column of Table II that as the activity level of the relationship diminishes from active, through intermediate, to passive, whether or not a social contact would be missed becomes an increasingly important way of discriminating between friends and acquaintances.

Latent-class analysis

Latent-class analysis (Lazarsfeld & Henry, 1968) has its origins in sociology and psychology and may be regarded essentially as a method of cluster analysis particularly suitable for categorical data. The basic model postulates an underlying categorical *latent* variable (Everitt, 1984) with, say, *c* classes; within any category of the latent variable the manifest (or observed) categorical variables are assumed to be independent of one another. Observed relationships between the manifest variables are thus assumed to result from the underlying classification of the data produced by the categorical latent variable.

The analysis was carried out on the 3769 social contacts of the patients with full SNS data using the same variables as in the log-linear modelling. The purpose was to discover whether the patients' social contacts fell into natural groupings, and to note the distinctive

TABLE III

Results of latent-class analysis of patients' social contacts: probability of positive response

Group	*No. of contacts*	*%*	*Missed*	*Friend*	*Confidant*	*Interaction*		
						Passive	*Intermediate*	*Active*
1	2027	54	0.11	0.01	0.22	0.74	0.19	0.07
2	388	10	1.00	0.86	0.47	0	0.20	0.79
3	1354	36	0.33	0.74	0.23	0.31	0.57	0.11

character of each grouping in terms of the SNS variables employed.

Results of latent-class analysis

The best-fitting solution was found to be one that assigned the named contacts to three groups. These are displayed in Table III, which gives the percentage of contacts falling into each group, and the probability that members of a group will rate positively on each of the four variables.

Group 1 is the largest, containing more than half the named contacts. They have low probabilities of being rated positively on any variable except passive interactions, that is, social interchange is limited to greetings. The pattern of rating that on common-sense grounds has least social significance consists of zeros on the missed, friend and confidant variables, and a passive rating on the activity variable. This pattern characterises 51% of the group 1 contacts, but by contrast only 3% of group 3 contacts, while it does not occur at all in group 2. It appears from these findings that group 1 consists of individuals whose names or roles are known to the patients, but contact with whom may have little or no social significance. We have, therefore, chosen to designate members of this group as 'ciphers'.

Group 2 is the smallest group, with no more than 10% of all contacts. They have the highest probabilities of being rated positively on the three dichotomous variables and of participating in active social interactions. It is noteworthy that its members have a 100% chance of being missed and no chance of being involved in passive interactions. The pattern indicating the most significant social relationship consists of a positive rating on the missed, friend and confidant variables, and an active rating on the activity variable. This pattern is shown by 32% of group 2, only 0.6% of group 3, and does not occur at all in group 1. It is evident that group 2 comprises people with whom the patients have the most intense social relationships.

Group 3 in many respects occupies the middle ground between the other two groups. Its members have relatively low probabilities of being missed or considered a confidant compared with group 2, but almost as high a chance of being viewed as a friend. The kind of social interaction that characterises this group is intermediate, namely the exchange of goods and services.

It can be seen from Table III that in each group a different type of social activity predominates. In group 1, interactions rarely go beyond simple greetings, in group 2 interchanges are mainly conversational, while in group 3 non-verbal transactions are the norm.

TABLE IV

Relationships between the activity dimension and designated friends

Nature of interaction	*No. of contacts with friends*	*% of all contacts at activity level*	*No. of contacts with others*
Active	1181	59	809
Intermediate[1,2]	352	48	378
Passive	242	23	807

1. Intermediate v. active, $\chi^2 = 26.6$, $P < 0.001$.
2. Intermediate v. passive, $\chi^2 = 122.9$, $P < 0.001$.

In view of these findings, it was thought to be potentially informative to explore the relationship between the three types of social activity and the designation of 'friend'. This analysis is presented in Table IV, from which it is evident that non-verbal transactions stand between conversations and simple greetings as indications of friendship. In fact they are considerably closer to active interactions in this respect than to passive ones.

Discussion

These analyses were performed with the aims of streamlining the SNS and of gaining a deeper understanding of the social relationships of long-stay patients. The best-fitting model to emerge from the log-linear analysis revealed that all four variables were included in either the first-order or second-order interactions. Hence each made a significant contribution to the variance, and none can be eliminated from the schedule. Although it is quite time consuming to administer to patients with extensive social networks, the informant soon learns the sequence of questions for each social contact and usually answers promptly.

The log-linear analysis also pointed to the salience of the confidant variable, which appeared in each of the three first-order interactions. Previous research on neurotic patients has highlighted the importance of confiding relationships (Brown & Harris, 1978; Henderson *et al*, 1978), which led to the inclusion of this variable in the SNS. Our data indicate that confiding plays an important part in the social relationships of long-stay patients, but there is one important difference from neurotic patients. Our subjects designated 14% of intermediate relationships as confiding. This intuitively surprising result is amplified by the latent-class analysis and is discussed below.

The findings from the latent-class analysis were most intriguing, since none of the dichotomous variables included in the SNS discriminated clearly between the naturally occurring groups. In constructing the schedule, we had included the variables friend and confidant with the expectation that they would be invested with a similar significance for the patients as for ourselves. This is evidently not the case, since members of groups 1 and 3 are equally likely to be designated as confidants. This is particularly surprising for group 1, for whom the great majority of interactions are confined to simple greetings or non-verbal interchanges. From our viewpoint it is difficult to envisage confiding in someone in the absence of a modicum of conversation. This could engender doubt about the reliability of the patients' use of the term confidant. On the other hand it would be incautious to assume that long-stay patients share our perceptions of social relationships. It is conceivable that for patients with poor conversational skills and restricted expression of emotion, the exchange of goods and services creates as intimate a bond as the sharing of confidences.

In view of the unexpected results from the latent-class analysis, it is our intention to repeat it on SNS data collected from discharged long-stay patients living in the community to determine whether the same groupings emerge.

Whereas friends are virtually non-existent among the ciphers of group 1, there is almost as high a probability of being designated a friend in group 3 as in group 2. It was a surprise to us that being 'missed' proved to be the best discriminator out of the dichotomous variables, applying to every member of group 2, but only a third of group 3. These findings suggest that the criteria used in everyday life outside of psychiatric institutions to discriminate between different intensities of social relationships are not being employed to the same extent by these long-stay patients. Inspection of Table III suggests that instead they are depending more on the quality of social interaction. In each group, a different mode of interaction predominates. In group 1, the ciphers, it is unusual for social interchange to extend beyond a greeting. In group 2 there is a nearly 80% chance of regular conversations occurring, while group 3 is characterised by non-verbal transactions. We interpret this to mean that for long-stay psychiatric patients, a conversation with another person is of much greater social significance than it would be for the man in the street. Participation in conversations seems to indicate a relatively high degree of social bonding between patients. Morgan (1979) attested to the difficulty that healthy people experience in maintaining conversations with long-stay patients. He reported that one patient did not "know the name of the man he chose as his companion all the week; he looked on it as a piece of unnecessary knowledge since they never spoke to one another". As a corollary to the relative paucity of conversation, non-verbal exchanges of goods and services loom much larger in establishing the social relationships of these patients than they do in life outside institutions. Part of the basis for this assertion derives from the observations made by Dunn *et al* (previous paper, this issue, pp. 842–848). Furthermore we found that non-verbal transactions were significantly stronger indicators of friendship than greetings, and were actually closer to conversations in this respect (see Table IV).

In seeking terms by which to refer to groups 2 and 3, we came to realise that no suitable words existed. This is because language develops to express common experiences and needs, and the social relationships of long-stay patients are out of the ordinary. There is no word in common parlance to designate a social relationship based on non-verbal transactions. We have chosen to coin the term 'transactors' for members of group 3. In the same vein, we propose to call members of group 2 'conversors'.

These findings have implications for the resettlement of long-stay patients in the community. Of the 489 patients with SNS data, 47 (9.6%) had networks consisting only of people with whom they had passive interactions (ciphers). These patients correspond to the 'asocial' observed in the patients' club by Dunn *et al.* The total number in the hospital population is likely to be considerably higher, since we found that those refusing to complete or incapable of completing an SNS were more socially inept than the others. The question of whether staffed group homes with high proportions of asocial patients are viable was raised in the previous paper.

A slightly higher proportion of the patients with SNS data (12.1%) had some intermediate interactions, but no active ones. These patients depend on non-verbal transactions to sustain social relationships. While our observations have shown that these are an integral feature of the patients' club, where tea and cigarettes are on sale, it is questionable whether such activity is feasible in staffed group homes, the commonest form of reprovision for Friern and

Claybury patients. If not, then it is possible that drop-in centres in the community supply a suitably conducive atmosphere for the exchange of goods and services among patients, and the establishment of social bonds. Observational studies in these settings are called for.

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The Social Functioning Scale

The Development and Validation of a New Scale of Social Adjustment for use in Family Intervention Programmes with Schizophrenic Patients

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Social functioning as an outcome variable in family interventions with schizophrenic patients has been a relatively neglected area. The requirements of a scale of social functioning to measure the efficacy of family interventions include: the measurement of skill/behaviour relevant to the impairments and the demography of this group; the ability to yield considerable information with an economy of clinical time; and the establishment of 'comparative' need through comparison between subscales and with appropriate reference groups. Results from three samples show that the Social Functioning Scale is reliable, valid, sensitive and responsive to change.

Within the last ten years, significant advances in the management of schizophrenia have come from studies of psychosocial intervention in the family environment in which the patient resides. These studies have largely focused on relapse as the major outcome variable; where social functioning has been measured, the results are equally favourable (Falloon *et al*, 1984).

This focus on relapse is surprising since impairment of social functioning is widespread in schizophrenia and may reflect a primary impairment as well as a secondary disability (Bellack *et al*, 1990). Deterioration in interpersonal relationships forms part of the defining characteristics of the syndrome and withdrawal and impairment in life-role functioning (social/recreation activity, independence/daily living skills, etc.) are listed as residual symptoms in DSM–III–R (American Psychiatric Association, 1987). The family interventions of Tarrier *et al* (1988) and Birchwood & Smith (1987) have directly targeted the raising of social functioning. These interventions require a comprehensive assessment of social functioning in which a direct comparison is made *between* its different components, and relative to established norms, in order to identify an individual's strengths and weaknesses.

The methods of assessing social adjustment have been reviewed by Weissman (1975, 1981). She indicates (1975, p. 1251) that many of the available scales have limitations for use with chronic disorders such as schizophrenia since this group may not be functioning in the roles that are assessed (e.g. current work, marital and parental roles). She advises that assessment of social functioning in a schizophrenic population should therefore assess more 'fundamental' characteristics, such as level of independence (competence and performance), social engagement/withdrawal, friendships/interpersonal functioning, and daily activities.

One particular limitation of available scales for family intervention is that they require a normative judgement by raters; for example, the Social Behaviour Schedule of Sturt & Wykes (1986) rates behaviour in terms of severity of problem. As Platt *et al* (1980) have pointed out, norms vary with characteristics such as age, sex, employment status, presence of disability, etc., and may lack external validity. In the case of schizophrenic patients who continue to live with their families, an unusual and difficult normative judgement about social adjustment will be required since this will refer to a 'disabled' group that will be largely unemployed, male and single. Furthermore, the judgements required in some scales refer to the presence of problems rather than strengths.

One further problem of many of the available scales for family interventions is the use of what can be a lengthy interview requiring trained raters (e.g. the SBAS; Platt *et al*, 1980). Those clinicians interested in implementing family interventions in a service will regard the investment of time required as excessive in the context of what is certain to be a treatment demanding of resources (Smith & Birchwood, 1990).

The ideal characteristics of a social adjustment scale for use with family interventions may therefore be summarised as follows:

(a) comprehensive and permitting comparisons between subscales
(b) sensitive to the likely level of impairment and relevant to the community tenure of this group
(c) independent of the normative judgement of 'independent' raters

(d) norms available for a population with a comparable age, sex and (un)employment structure
(e) able to yield considerable information with an economy of clinical time
(f) available in self-report or informant versions
(g) conforming to accepted criteria of reliability, validity and sensitivity.

The Social Functioning Scale (SFS) was constructed specifically to tap those areas of functioning that are crucial to the community maintenance of individuals with schizophrenia. In this respect, the content of the SFS was informed by the areas focused on in the successful psychosocial intervention programmes of Hogarty *et al* (1979), Paul & Lentz (1977), Stein & Test (1980), and the impairments and disabilities assessed by the Disability Assessment Schedule (World Health Organization, 1980). The seven areas selected are shown in Table IV and include:

(a) social engagement/withdrawal (time spent alone, initiation of conversations, social avoidance)
(b) interpersonal behaviour (number of friends/heterosexual contact, quality of communication)
(c) pro-social activities (engagement in a range of common social activities, e.g. sport)
(d) recreation (engagement in a range of common hobbies, interests, pastimes etc.)
(e) independence-competence (ability to perform skills necessary for independent living
(f) independence-performance (performance of skills necessary for independent living)
(g) employment/occupation (engagement in productive employment or structured programme of daily activity).

The SFS was designed with two requirements in mind. The first was to provide a detailed assessment of strengths and weaknesses, both to guide an intervention and to provide the clinician with some possible specific goals, subject to negotiation with the individual and relatives. Thus it was intended that the SFS would establish 'comparative' need (Bradshaw, 1972) based on comparison with reference groups as distinct from 'felt' or 'expressed' need (which requires negotiation/discussion with the individual) or action-based needs assessment (Brewin *et al*, 1987). The second requirement was the ability to synthesise such detailed coverage into coherent, reliable continua.

Assessing personal and social functioning is not straightforward. Some measures have assessed role functioning and require judgement about the extent to which an individual fulfils a social role (e.g. worker, parent). As indicated above, these assessments require a normative judgement, which may prove unreliable. The SFS uses a different approach by enumerating basic skills, social behaviour, etc. which informants record as present or absent, thereby avoiding 'evaluative' decisions. In this respect, the SFS has some similarities with the 'MRC Needs for Care Assessment' which was developed for the long-term mentally ill in residential settings (Brewin *et al*, 1987). The SFS also distinguishes lack of competence from lack of performance: lack of competence refers to the absence or loss of a skill; lack of performance refers to non-use or disuse of an available skill. This distinction was measured solely in relation to skills necessary for independent living, as it was felt that informants would have difficulty in achieving this distinction in other areas (e.g. social skills v. social performance). The SFS was developed by Birchwood (1983) and underwent extensive development through psychometric analysis before the final version was established.

The present study examined the reliability, validity, sensitivity and utility for family interventions of the SFS.

Method

Several distinct groups of subjects were recruited at different stages of this study.

A sample of 334 schizophrenic out-patients (Table I) all with a clinical diagnosis of schizophrenia and conforming to the broad CATEGO 'S' class including S+, S?, P+ and O (Wing *et al*, 1974) was a compendium taken from previous and ongoing research studies (Birchwood, 1983; Birchwood *et al*, 1989; Smith & Birchwood, 1987, 1990). Each of the samples of which this is a compendium were defined on the same clinical criteria, had been living (or were in daily contact) with their relative from the first or second episode, and were drawn from the same catchment area. All patients were in contact with the mental health services by virtue of their attendance at out-patient clinics, or referral to a community psychiatric nurse or the clinical psychology services. It should be noted that this sample was taken during a period of investigation into families' service needs (Smith & Birchwood, 1990), when the large majority of patients living with their families known to the service were approached. In the total sample 88% were taking oral or depot neuroleptics, and 24% were in productive paid employment. In keeping with other studies (e.g. Tarrier *et al*, 1988), the sample contained an excess of males. Two subgroups of this sample took part in reliability analyses: 30 sets of parents completed the SFS independently about their schizophrenic offspring; in a further 25 cases, relatives' data were compared with the SFS completed by 25 symptom-free patients.

A sample of 100 normal subjects were recruited via their relatives. Relatives were approached by interviewers in 'key sites' throughout the catchment area from which the patient sample was drawn (e.g. shopping and job centres), and requested to complete the SFS about an offspring or relative with whom they were in close contact. Interviewers were instructed to suggest a male relative or offspring in two out

TABLE I
Demographic characteristics of the samples

	Schizophrenic	*Community*	*Matched groups*	
			Schizophrenic	*Siblings*
Number	334	100	59	59
male	238	65	47	36*
female	96	35	12	23
Mean age (s.d.): years	30.8 (10.1)	30.5 (8.4)	29.6 (8.4)	30.5 (9.6)
Parental social class: %				
professional, clerical	39	38	68	68
skilled manual	33	39	20	20
semiskilled, unskilled manual	15	10	7	7
unemployed	13	13	5	5
Mean no. of previous admissions (s.d.)	3.2 (3.1)	–	3.7 (2.7)	–
Mean illness duration (s.d.): years	8.8 (7.4)	–	8.2 (8.2)	–

*$P<0.03$.

of every three interviews. The resulting sample had a similar sociodemographic distribution to the schizophrenics but somewhat more (60%) were employed.

In a further group, 59 relatives were requested to complete the SFS about their schizophrenic offspring. Thirty patients conformed to the above diagnostic criteria; a further 29 had a clinical diagnosis of schizophrenia and were recruited through the National Schizophrenic Fellowship. Each relative completed the SFS in respect of another, non-schizophrenic offspring, where possible of the same sex and similar age. Table I shows that the matched groups were of similar age but as the circumstances of sampling selected for males, the siblings had a greater proportion of females.

In order to study the relationship between SFS status and the presence of positive and negative symptoms, a subsample of 53 schizophrenics from the main sample (mean age: 24.2 years; mean illness duration 2.2 years) were administered the Present State Examination (PSE; Wing *et al*, 1974).

TABLE II
Reliabilities and intercorrelations of the SFS

	Full scale	*Withdrawal*	*Interpersonal*	*Prosocial*	*Recreation*	*Independence–competence*	*Independence–performance*	*Employment occupation*
No. of constituent items	7	5	5	23	15	13	13	
Mean inter-item correlation	0.44	0.37	0.36	0.29	0.25	0.35	0.33	–
Mean item–total correlation	0.71	0.49	0.40	0.37	0.30	0.55	0.53	–
Reliability: alpha	0.80	0.72	0.71	0.82	0.69	0.87	0.85	–
Inter-rater reliability ($n=30$)	0.94	0.96	0.88	0.69	0.82	0.93	0.91	0.96
Rater self-report correlation ($n=25$)	0.78	0.62	0.69	0.63	0.65	0.69	0.70	0.99
Correlation with:								
Social engagement/ withdrawal	0.67							
Interpersonal	0.70	0.39						
Prosocial	0.75	0.47	0.63					
Recreation	0.71	0.29	0.43	0.52				
Independence–competence	0.63	0.42	0.27	0.23	0.39			
Independence–performance	0.80	0.48	0.41	0.45	0.58	0.61		
Employment	0.72	0.46	0.38	0.48	0.42	0.33	0.61	–

Results

Reliability

The main results of the reliability analyses are shown in Table II.

Four aspects of reliability were studied. The coefficient alpha (Guttman, 1945) is based on a single administration of the test and rules out the possibility that any real changes in social functioning appear as low reliability, as might occur with the test–retest method. The alpha coefficients in Table II are uniformly high (a reliability coefficient when multiplied by 100 expresses directly the percentage of test score variance attributable to 'true' variance in the characteristic being measured). Inter-rater and rater self-report reliabilities confirm this impression and suggest that the SFS is measuring characteristics about which both the individual and relatives concur. No difference in SFS scores between rater and self-report was observed. Finally, since the SFS scales are obtained through summation of constituent items, it is desirable that part of the variance in response to individual items should be determined by the characteristic measured by the total scale (Cochrane, 1980). Item–total correlations for the scales show there is a high level of internal consistency in the SFS scales. The pro-social and recreation scales are somewhat weaker in this respect as might be anticipated, as these are not 'traits' in the usual sense but a compendium of activities.

Validity

Two methods of establishing validity were used: construct validity and the criterion group method.

With regard to construct validity, the question posed was whether the SFS scales were connected via a common factor or construct ('social adjustment'). Accordingly, a factor analysis was performed using the alpha method of factoring (Harman, 1967). This method was chosen as the variables were considered a sample from the universe of variables which might conceivably relate to the concept 'social adjustment'. After iterations, one single factor was extracted with an eigenvalue of 3.96, accounting for 57% of the variance. The factor loadings (Table III) represent the actual correlation between each item and factor scores; these are both uniform and high. Since this was undertaken with the schizophrenic ($n=334$) and normals ($n=100$) combined, and since it is possible that these two groups are at least quantitatively different in social functioning, this factor structure might be distorted. Two further factor analyses were undertaken within these two groups (Table III). These revealed a similar although marginally weaker factor structure. Interestingly, within the community group, there was no loading on independence (competence) as there was little variation on this scale for this group. This result, together with the high intercorrelation between the SFS scales, suggests that it is appropriate to obtain a mean score of the SFS scales ('full scale' in Table I).

As the scales have differing means and variances, each scale was standardised and normalised using a '*T*' transformation to a mean of 100, standard deviation of 15, using the unemployed schizophrenic group as the reference population (see below). The reliabilities for the 'full scale' (constructed after the transformation) are shown in Table II. The construction of the 'full scale' after transformation in effect assigns the subscales equal importance. Although there was no reason *a priori* to assume that they were not of equal importance, the substantial intercorrelation between subscales, the high average (corrected) subscale–full-scale correlation, and the extraction of a single strong factor loading uniformly across subscales all suggest that they are. There may of course be compelling *clinical* reasons to 'weight' certain subscales in the light of individual circumstances; in this sense the SFS requires careful interpretation, in keeping with other scales.

TABLE III
Factor analysis of the SFS scales

	Factor loading		
	All subjects (n = 434)	*Schizophrenic (n = 334)*	*Community (n = 100)*
Social engagement/ withdrawal	0.76	0.63	0.80
Interpersonal	0.76	0.65	0.62
Independence–competence	0.69	0.62	0.04
Independence–performance	0.78	0.75	0.62
Recreation	0.76	0.60	0.71
Pro-social	0.79	0.65	0.71
Employment	0.72	0.60	0.63
Eigenvalue	3.90	3.48	3.33
% variance	57	49	41

The criterion groups were as follows. The schizophrenic group was contrasted with the community sample as it was anticipated that the schizophrenics should have lower scores in view of their well documented social impairments (World Health Organization, 1980). As these groups showed different frequencies of unemployment, employment/unemployment was extracted as a factor in a group × employment factorial ANOVA. The results (Table IV) revealed that these criterion groups are distinguished to a highly significant degree across all SFS scales.

Of particular interest was that the interaction of schizophrenia and unemployment is not a pernicious one in terms of social functioning (Table IV column 3). The significant interaction on the recreation variable was due to an elevation in recreation scores among the employed normals ($P<0.01$). The significant interaction on the employment scale arises because unemployed normals were rated as being in more active pursuit of work compared with unemployed schizophrenics ($P<0.05$).

The contrast between schizophrenics and their siblings provides a further validity check (but keeps 'rater' and 'environment' constant). Table IV shows that the groups are discriminated on all SFS scales to a highly significant degree.

Descriptive statistics for the criterion groups on the full-scale SFS (Table V) show the high degree of discrimination achieved.

TABLE IV
ANOVAS for the main groups on the eight SFS scales

	Schizophrenic v. normal (d.f. 1,406) *F*	*Employed v. unemployed (d.f. 1,406)* *F*	*Interaction: schizophrenic × employment (d.f. 1,406)* *F*	*Schizophrenic v. sibling (d.f. 1,117)* *F*
Social engagement/withdrawal	37.5*	58.8*	<1	96.0*
Interpersonal	52.5*	27.3*	<1	81.4*
Pro-social	98.8*	37.9*	2.0	64.3*
Recreation	60.5*	48.2*	11.5*	84.3*
Independence-competence	54.6*	6.6***	2.9****	68.5*
Independence-performance	31.3*	18.5*	<1	78.7*
Employment	<1	118.4*	5.0**	86.8*
Full scale	95.6*	100.6*	<1	119.3*

*$P<0.001$, **$P<0.025$, ***$P<0.05$, ****$P<0.1$

TABLE V
Means, s.d.'s and confidence intervals of the main contrast groups on the mean SFS

	Schizophrenic		*Non-schizophrenic*		*Matched groups*	
	Employed	*Unemployed*	*Employed*	*Unemployed*	*Schizophrenic*	*Siblings*
No. of subjects	80	254	60	40	59	59
Mean full-scale SFS score	111.3	100.0	124.6	112.2	102.2	123.8
s.d.	9.1	10.0	5.0	7.6	12.5	6.6
95% CI	109–114	98–102	123–126	110–115	99–106	122–126

The relationship between social functioning and positive/negative symptoms was studied in the subsample of 53 patients referred to above. Patients were assigned to a 'non-negative' group if they scored '0' on 'blunted affect', 'loss of interest' and 'withdrawal'. Subjects scoring 1 or more on one or more of these items were assigned to a 'negative' group. The point-biserial correlation coefficient between presence/absence of negative symptoms and full-scale SFS was $r=0.44$ ($P<0.01$). A 'positive' and 'non-positive' group was similarly identified based on the presence of hallucinations, delusions and disorders of thinking (thought insertion, echo, etc.). The point-biserial correlation with mean SFS was $r=-0.46$ ($P<0.01$). The multiple correlation of positive *and* negative symptoms with SFS was $R=0.55$.

Sensitivity

Sensitivity refers in part to the extent to which a scale can respond to differences in the characteristic being measured. This is an important feature of the SFS since it is designed to be used as a continuous measure (as opposed to a way of identifying 'cases'). One indirect method of assessing sensitivity is via the distribution and range of scores on the SFS. Table VI shows the distribution of scores for the community sample and the schizophrenic main sample. This shows a considerable range for the schizophrenic group; the community group showed a distribution around a higher mean with a moderate positive skew (both groups passed the Kolmogorov–Smirnov 'goodness-of-fit' test for a normal distribution). It is of interest to note that scores in excess of 115 are occupied by 74% of the community sample in contrast to 14.4% of the schizophrenics.

Sensitivity to change cannot be inferred from sensitivity to individual differences however. Evidence for sensitivity to change comes from a study of expressed emotion (EE) and family intervention, by Barrowclough & Tarrier (1990). They report significant elevations in SFS scores in their high-EE intervention group and low-EE control group but not the high-EE control group. These changes were in line with changes in relapse. They also report that the 64 patients from the high-EE group had significantly lower scores on

TABLE VI
Frequency (%) of scores on the mean SFS scale for schizophrenics and non-schizophrenics

SFS score	*Schizophrenics (n = 334)*	*Community (n = 100)*
55– 65	0.3	0
66– 75	0.3	0
76– 85	5.0	0
86– 95	21	1
96–105	30	6
106–115	29	19
116–125	13	42
126–135	1.4	32

the SFS than the 19 in the low-EE group on the two independence scales, the recreation scale, and the full-scale score. Similar differences have been reported elsewhere (Brown *et al*, 1972).

Discussion

The study has provided strong support for the reliability and validity of the SFS. In terms of reliability, the SFS itself gives rise to minimal measurement error as shown by the high internal reliabilities (coefficient alpha) and that both informants and patients concur in their observations. Regarding validity, the criterion groups were strongly differentiated and SFS scores correlated with the presence of both negative ($r = -0.44$) and positive ($r = -0.46$) symptoms. It is generally accepted that negative symptoms contribute to deficits in social functioning (e.g. Wing & Brown, 1990; Bellack *et al*, 1990); however, the correlation with positive symptoms was considered unusual. Data from two well known follow-up studies seem in fact to confirm this finding. Strauss & Carpenter (1977) in their five-year follow-up of schizophrenics in the International Pilot Study of Schizophrenia (World Health Organization, 1979) report a significant correlation between positive symptoms and quantity and quality of social contacts ($r = 0.62$) and employment ($r = 0.47$). Shepherd *et al* (1989) report that the presence of first-rank symptoms after five years was strongly associated with depressed social outcome using a χ^2 analysis ($P < 0.001$).

These results then show that the SFS fulfil essential psychometric criteria (Weissman, 1981). These data notwithstanding, what assurance is there that the SFS is measuring the human characteristics it purports to?

First, each scale has overwhelming face validity: the recreation scale inquiries about common recreations; the independence scale inquiries about the ability/opportunity to perform daily living skills; the social engagement/withdrawal scale inquiries about social avoidance, and so on. The scales themselves are detailed and sample widely within each characteristic. Second, the high internal coherence of these scales (item–total correlations) suggests that the scale totals reliably summarise the concepts contained in each. Third, the factor analysis yielded a single, powerful factor accounting for nearly 60% of the variance. This factor loaded uniformly and strongly across the constituent scales. It is concluded, therefore, that this factor corresponds to a generalised construct best described as 'psychosocial functioning'. The overall full-scale score, which gives equal weight to constituent scales, has a close correspondence with this statistical factor, thereby adding weight to its validity.

The SFS is intended to measure a continuous characteristic; it is clearly able to discriminate criterion groups but its ability to discriminate lesser differences is crucial. In other words, are two individuals with different SFS scores of differing social adjustment? The wide range of (normally distributed) scores and the observation that individuals and informants are clearly making reliable and fine discriminations in their social behaviour (about which they concur) would indicate that different SFS scores are associated with perceived differences in social behaviour and skill.

The SFS has certain advantages for the clinical setting in that it requires little professional time, it is acceptable to patients and their families, and it yields a great deal of information about an individual's abilities and activities which can be summarised and interpreted through the derivation of the scales. Comparisons may be made between the scales and relative to the norms of a comparable community group, to identify strengths and weaknesses. The SFS has been employed in this way by the authors in their family intervention study (Birchwood & Smith, 1987) and also by Barrowclough & Tarrier (1990). As indicated above, Barrowclough & Tarrier (1990) found that the SFS was responsive to change (in line with improvements in relapse) and discriminated the EE groups.

It is concluded that the SFS is a reliable, valid and sensitive measure of social functioning relevant to the impairments and needs of individuals with schizophrenia and of use to researchers and clinicians concerned with this variable in family (or indeed other psychosocial) interventions.

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Comparison of the General Health Questionnaire and the Hospital Anxiety and Depression Scale

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The specificity and sensitivity of the HAD, 12-item GHQ and CIS were calculated by comparing the scores of dermatological patients on these tests with a criterion measure of disorder. Since psychiatry, along with many other branches of medicine, does not have an error-free criterion, it was assumed that the criterion was an underlying latent construct which was measured by all of the tests and could be derived by factor analysis from the scores on them. No differences were found between the two questionnaires (HAD and GHQ) in their ability to detect cases of minor psychiatric disorder although they were somewhat less reliable than the CIS.

It is commonplace to study the validity of simple tests by comparing them with a 'gold standard' or criterion and thus calculating the sensitivity and specificity. These validity indices thus give an estimate of the accuracy with which a test measures the criterion.

Unfortunately psychiatrists, along with many of their medical colleagues, do not have a criterion measure of disorder. All the currently used standardised interviews, which are traditionally regarded as the criterion, are not free of error and show modest repeatability. Indeed, it is ironic that the sensitivity and specificity of the more repeatable General Health Questionnaire (GHQ; Goldberg, 1972) is calculated in relation to the less repeatable standardised interviews. When the criterion shows error, the sensitivity and specificity of the test will apparently be reduced. Furthermore, conventional statistics only estimate sampling error and do not account for variation resulting from error in the criterion.

The main aim of this paper is to use factor-analytic models as a form of validity testing in addition to the traditional method of comparing a questionnaire with a standardised interview. Such models assume that the criterion is an underlying construct (in this case, overall severity of minor psychiatric illness) which is measured by the questionnaires and interviews and can be derived by factor analysis from the scores on them.

Screening for psychiatric disorder in the physically ill

There has been much debate about the validity of using self-report questionnaires in the physically ill (Kirk & Saunders, 1979; Mayou & Hawton, 1986; Goldberg & Williams, 1988) and other authors have suggested that the GHQ score used to define a 'case' should be raised in those with neurological conditions (Bridges & Goldberg, 1986) or angina (Vasquez-Barquero *et al*, 1985). This is usually attributed to the overlap between the somatic symptoms of psychological disorder and those of physical disease. False positives are also explained by questions in the GHQ assessing social functioning, for instance "Have you recently been getting out of the house as much as usual?" The influence of physical illness on assessments of psychiatric disorder is a potential source of bias in much psychiatric research.

Structured interviews partly rely upon the interviewer (usually a psychiatrist) making judgements based on clinical impression. This could lead to an underestimate of the prevalence of psychiatric disorder in the physically ill. Firstly, symptoms (e.g. fatigue, insomnia) may be attributed to physical illness, or omitted from the score through caution, although in fact they are caused by psychological illness. Secondly, the emotional consequences of physical illness could be viewed as a normal or understandable response to a life-threatening diagnosis rather than a psychiatric disorder. Both these factors would reduce the apparent prevalence of psychiatric disorder in the physically ill and lead to an increase in the proportion of false positives. Again, the absence of a criterion prevents an easy solution to this problem.

Zigmond & Snaith (1983) devised the Hospital Anxiety and Depression Scale (HAD) specifically for detecting anxiety and depression in the physically ill, and it is therefore of interest to compare the GHQ with the score derived from combining the two scales of the HAD. The main differences between the GHQ and the HAD are that the latter asks only about psychological symptoms and does not have the 'same as usual' option, a response which scores zero in the GHQ.

The HAD has been compared with clinical rating scales (Zigmond & Snaith, 1983; Aylard *et al*, 1987) and standardised interviews (Barczak *et al*, 1988; Andrews *et al*, 1988) but only two papers have compared the HAD with the GHQ. Wilkinson & Barczak (1988) compared the HAD with the GHQ-28 in a general-practice sample, using the structured clinical interview for DSM-III as the criterion. Unfortunately there was no indication whether the slight superiority of the HAD could be attributed to chance. Aylard *et al* (1987) also compared the HAD with the GHQ-28 but like Zigmond & Snaith (1983) they did not use a single threshold to define a case and so their studies produced inflated values for sensitivity and specificity by excluding doubtful cases.

Using factor-analytic models, this study compared the HAD, the 12-item GHQ and the Clinical Interview Schedule (CIS; Goldberg *et al*, 1970), in their ability to detect cases of minor psychiatric disorder in a sample of new referrals in a hospital setting. Using this method the two self-report questionnaires could be compared with each other and with the CIS.

The study was conducted in a dermatology clinic and dermatological conditions do not usually lead to impaired social functioning. There is little empirical work on patients with dermatological disease and although these disorders are reputed to frequently arise psychogenically there is no quantitative evidence that this is so. Wessely & Lewis (1989) concluded that there is little evidence to suggest either that dermatology patients have an excessive prevalence of psychiatric conditions or that there is an obvious link between particular skin disorders and psychiatric morbidity. The 12-item GHQ was chosen partly because it has a similar number of items to the HAD (14 items) and also because it contains no somatic symptoms. Goldberg & Williams (1988) concluded, on the basis of an extensive review and meta-analysis, that the validity of the GHQ-12 is comparable with that of the longer versions of the GHQ.

Method

On each day selected for study, a random sample of all new patients attending the Dermatology Out-patient Clinic of King's College Hospital were invited to take part in the study. Each subject was asked to complete the HAD and 12-item GHQ and to be interviewed by one of the authors using the CIS. This was part of a larger study where other information was collected and these results have been presented elsewhere (Wessely & Lewis, 1989).

A 'case' was defined if the subject scored 13 or more on the total weighted score of the CIS (Goldberg *et al*, 1970). Relative operating characteristic (ROC) curves were fitted using the ROCFIT program (Metz *et al*, 1984).

The GHQ was scored using the traditional method (0, 0, 1, 1; Goldberg, 1972) except for analyses in which factor analysis was used. In these cases the Likert (0, 1, 2, 3) method of scoring was adopted. This method leads to a distribution of scores which more closely approximates the normal distribution.

Use of factor-analytic measurement models

The sensitivity, specificity and kappa values of a self-report questionnaire, traditionally assessed by comparison of the questionnaire with a standardised interview (the criterion), may be reduced as a result of error in the criterion as well as that in the test itself. This should not affect the comparison of two tests except that conventional statistics only estimate sampling error and so apparently statistically significant differences between tests should be interpreted cautiously. One way of attempting to circumvent this problem is by use of factor-analytic measurement models (Allen & Yen, 1970; Joreskog, 1971). The model assumes that all three psychiatric measures used here are measuring the same underlying construct which can be represented by a single factor derived by maximum-likelihood factor analysis from the scores on the three tests. All three measures can then be compared with the factor which is used as the criterion. In this study the LISREL VI program was used (Joreskog & Sorbom, 1974).

Two analyses can be performed using this method. Firstly, the reliabilities of the scales can be calculated using LISREL VI. Assuming that all variation apart from the factor is random error, LISREL can calculate the variance of this random-error term. From classical test theory, the reliability is given by the variance of the true score divided by the total variance (Carmines & Zeller, 1979). The variance of the factor is used as the estimate of the true variance and the total variance is therefore calculated by adding the factor variance to the variance of the random-error term calculated using LISREL. This estimate of reliability can be conceived as the accuracy with which each test measures the factor-derived construct.

The second analysis performed determines a threshold score on the factor and this is used instead of the CIS to define a 'case'. In this study the threshold on the factor was determined using ROC analysis (Mari & Williams, 1985). All three measures can then be compared with the factor-derived case definition.

In summary, the assumptions underlying the measurement model are as follows:

(a) the two questionnaires and the total weighted score of the CIS are measuring the same psychological construct
(b) this construct can be represented by a single factor derived by maximum-likelihood factor analysis from the three measures
(c) each measure can be represented by a linear combination of the factor and a random-error term;

Table I

Comparison of: (a) the HAD with the GHQ when the CIS is used as the criterion; and (b) the HAD, GHQ and CIS when the presumed underlying factor score is used to define a case

		Se%	*Sp%*	*Bias*	*Kappa*	*s.e.*	*ROC area*	*s.d.*
(a)	HAD	72.3	77.1	1.06	0.490	0.082	0.88	0.033
	GHQ	78.7	77.5	1.13	0.550	0.078	0.84	0.046
(b)	CIS	89.8	95.6	0.96	0.859	0.048	0.98	0.012
	GHQ	85.7	85.3	1.06	0.704	0.066	0.91	0.032
	HAD	77.6	82.4	1.02	0.597	0.075	0.92	0.026

1. Se% = sensitivity.
2. Sp% = specificity.

errors in the different measures are assumed to be uncorrelated

(d) the test scores are assumed to be normally distributed.

Results

The sample consisted of 173 subjects of whom 117 (68%) completed the HAD, GHQ and CIS. Some data were collected on almost all the 56 non-responders. They did not complete all the measures, either because the experimenters could not contact them or they claimed they could not afford the time. The average age of the non-responders ($n=53$) was 45 years and of the responders 40 years ($t=0.3$, d.f. 166, $P>0.5$). Of the 56 non-responders, 55% were women while 60% of the responders were female ($\chi^2=0.35$, $P>0.3$). Of the non-responders, 35% (9/26) were 'cases' defined by the 1/2 cut-off on the GHQ-12, while 44% of the responders were cases using the same criterion ($\chi^2=0.84$, $P>0.3$). These results indicate that the group on whom complete data were collected was fairly representative of the original random sample.

The CIS suggested that the prevalence of minor psychiatric disorder was 40.2% (47/117; 95% CI 35.7–44.7%).

The mean score on the HAD anxiety scale (HADA) was 6.9 (s.d. = 4.23) and on the HAD depression scale (HADD) was 3.9 (s.d. = 3.41). The correlation between the CIS score and the HAD score (the sum of HADA and HADD) was 0.79, between the CIS and the GHQ was 0.76 and between the GHQ and HAD was 0.75.

Comparison of questionnaires using the CIS

Using the CIS-defined case as the criterion, the GHQ and HAD were compared. The recommended cut-off score for the 12-item GHQ is 1/2 (i.e. two or more is a case) but there is no accepted score for the HAD, although Wilkinson & Barczak (1988) and Andrews *et al* (1988) have suggested a 7/8 threshold. ROC analysis was used to define the best HAD cut-off (Mari & Williams, 1985), and in this sample it was 10/11. Using the sample to define the cut-off in this way tends to enhance the sensitivity and specificity of the HAD.

The sensitivities and specificities of the two questionnaires compared with the CIS are given in the first two columns of Table I(a), and the test bias (the ratio of test:criterion prevalence) is shown in the third column. The areas under the ROC curves (sixth column) give a measure of the ability of a test to distinguish a case from a non-case and an area of 1.0 indicates perfect agreement. The two ROC areas were not significantly different ($z=0.87$; $P=0.4$) using the method of Hanley & McNeil (1983).

The kappa statistic (Fleiss, 1981) is a measure of agreement which corrects for chance. It was also calculated with standard errors for both questionnaires (Table I; fourth and fifth columns). The kappas were compared using a resampling or 'bootstrap' method. Based upon 250 bootstrap samples, the mean difference between the kappa for the HAD and the GHQ was 0.074 and the standard error was 0.089, indicating there was not a significant difference ($P=0.4$).

The results for the GHQ show a slightly superior sensitivity and kappa, although the ROC area for the GHQ is somewhat smaller. This apparent discrepancy is probably because the sensitivity, specificity and kappa are calculated using a single threshold while the ROC area is calculated using several thresholds. In this instance the difference between the ROC areas is not statistically significant, but, in general, the results of the ROC area calculated with a curve-fitting program is probably a better estimate of agreement between test and criterion than indices calculated using a single threshold.

In conclusion, it appears there is no substantial difference between the two questionnaires when they are compared with the CIS.

Comparison of questionnaires using measurement models

The reliabilities calculated using LISREL were 0.83 for the CIS, 0.72 for the GHQ and 0.74 for the HAD.

Using the factor scores to define a case led to the results shown in Table I(b). Again, there appeared to be few differences between the GHQ and the HAD, and the difference in ROC areas between the HAD and GHQ was not significant ($z=0.13$, $P=0.9$; Hanley & McNeil, 1983). However, the levels of agreement with the factor-derived criterion was lower for the two questionnaires than for the CIS, and there was a significant difference between the ROC areas for the GHQ and CIS ($z=2.3$, $P=0.02$).

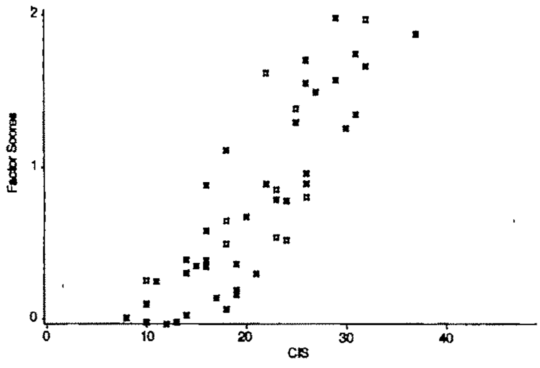

FIG. 1 Scores on the CIS for each individual plotted against the factor derived from the scores on the CIS, GHQ and HAD.

The total weighted scores on the CIS for each individual are plotted against the factor scores in Fig. 1. This illustrates that using the model, the CIS appears to be closely related to the construct represented by the factor, but is not free of error.

Discussion

In all the comparisons here, there were few differences between the HAD and the 12-item GHQ and they performed very similarly in this sample of dermatology patients. Comparison of the GHQ with other questionnaires, similar to the HAD in some respects, have found no differences in the ability of the various questionnaires to detect cases of minor psychiatric disorder (Goldberg *et al*, 1976; Mari & Williams, 1985).

Wilkinson & Barczak (1988) have argued that the HAD has a 'consistent threshold score' of 7/8. The difference with the present results (threshold 10/11) may reflect the different case definitions in the two standardised interviews, but it seems wise to avoid a fixed threshold for the HAD before it has been more widely studied.

In addition to the traditional approach to validity using a standardised interview as the criterion, factor-analytic measurement models were also used in an attempt to derive a better estimate of the underlying mental illness construct. The assumptions of the model, outlined earlier, are parsimonious and reasonable in the light of current psychiatric research into minor psychiatric morbidity. This was illustrated in Fig. 1. The analytic method seems to provide useful additional information to that obtained using the more established analyses.

In comparison with the differences between the two questionnaires, which were tiny, the measurement model suggested a small but definite advantage in the reliability of the CIS interview. This is reassuring, but not surprising as the CIS takes about ten times as long and contains many more questions than the GHQ or HAD. However, the difference in reliability is relatively modest and this questions the routinely made assumption that a standardised interview is free of error. It will be of interest to see whether interactive computerised assessments (Lewis *et al*, 1988), which can be longer and more thorough than pen and paper questionnaires, will also be more reliable.

The measurement model permits the calculation of the reliability of the observed variables in measuring the underlying construct. Reliability is a somewhat ambiguous term and the term repeatability has been recommended when the agreement between replicate measurements is estimated (Rose & Barker, 1978*a*). The circumstance here is quite different. The reliabilities are estimating the accuracy with which the tests are measuring the underlying construct, the presumed criterion. This method is therefore a form of validity testing, where the criterion is a continuous measure derived by factor analysis. Sensitivity and specificity measure the accuracy with which a test measures a categorical criterion. It has been persuasively argued that disease is best conceived as a continuum (Rose & Barker, 1978*b*) and this view seems especially appropriate for minor psychiatric disorders.

Conclusions

Measurement models have been used to try to overcome the problems of using standardised interviews as an error-free criterion. It seems that reasonable assumptions are needed for these models, and their use could provide additional insights into psychiatric measurement.

The results indicate that there were few differences between the HAD and GHQ in their ability to detect cases of minor psychiatric disorder in this setting. The standardised interview seemed to be somewhat more accurate than the self-report questionnaires.

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The Prediction of Rehabilitative Success after Three Years

The Use of Social, Symptom and Cognitive Variables

TIL WYKES, ELIZABETH STURT and RANDY KATZ

A three-year follow-up study of 49 psychiatric patients, representative of those found in long-stay care, tested whether behavioural, symptom and demographic variables as well as response-processing difficulties could predict levels of psychiatric care. The researchers were blind to the criteria for allocating patients to particular forms of care and the staff responsible for care had no access to information collected by the research team. Not only was it possible, using only these few factors, to predict the form of care, but one factor, response processing, was related to whether patients moved to more independent forms of psychiatric care.

There have been many studies investigating the prognostic significance of various factors in the outcome of psychiatric illness. Most of these studies have looked at acute populations or have carried out retrospective studies and are therefore of little use when trying to predict the success of chronic patients in a rehabilitation programme. It has become increasingly important in the past few years to identify such factors as further hospitals are due for closure and the move towards care in the community is implemented. Two recent studies, one in America and one in Britain, have suggested that several variables may be important. The American study laid great emphasis on chronicity, symptoms, social functioning and family history and also concentrated on a schizophrenic sample (Fenton & McGlashan, 1987). Our British study, although including many of the same variables, also included a novel set which measured cognitive difficulties (Wykes *et al*, 1990). A more representative group of chronic patients was also tested (i.e. a mixed diagnostic group). However, the study was only cross-sectional and further data need to be collected in order to test the predictive performance of the factors that we isolated. The present paper therefore reports data from a prospective three-year follow-up study.

Netherne Hospital, the base for this series of studies, has been designated a centre of excellence in rehabilitation, and although suffering the financial constraints placed on the National Health Service, it does have a number of different services in which to place people with psychiatric handicaps. These facilities are situated both in the hospital grounds and in the community. At the time of collecting data for this study, they consisted of a graded system of day- and night-care services which could be tailored to each patient's needs. Night care was divided into: patient's own home supported by a community psychiatric nurse, group homes, hostels, a series of villas with differing expectations of independence within the hospital grounds, and longer-stay traditional wards. Day care too was divided into a number of options including a sheltered workshop in the community, industrial therapy in the hospital grounds, occupational therapy either on the ward or in a special unit, and opportunities to take advantage of paid ward work. Because of the flexibility in the services to match patients' needs to the appropriate level of care, this hospital complex was seen as providing a suitable testing ground for the predictive success of different factors thought to be involved in rehabilitation potential.

In the previous cross-sectional study several variables were identified as likely to play a role in future service use. These fell into four categories:

(a) basic demographic data, such as highest level of employment, level of independent living, chronicity of illness
(b) current symptoms from the Present State Examination (PSE; Wing *et al*, 1974), such as level of positive or negative symptoms
(c) social behaviour problems, derived from the Social Behaviour Schedule (SBS; Wykes & Sturt, 1986)
(d) response-processing problems, derived from a choice reaction-time task fully described by Wykes *et al* (1990) (several reaction-time variables were related to care but the one of most interest is a measure of the difficulties patients have when a load is placed on the response-processing stage of Sternberg's (1969) information processing model).

Method

Forty-nine people from the cross-sectional study were chosen randomly from the Netherne psychiatric services and data were collected on the following schedules: basic demographic data, the PSE, the Social Behaviour Schedule, and choice reaction-time task. At the time of the initial survey, the subjects were situated in a wide range of facilities, but generally they fell into three main groups: those working in a community sheltered workshop and living in their own homes or a high-expectation villa in the hospital; those working in an industrial therapy unit and living in a hospital villa; and those living in a traditional long-stay ward with either no occupation or occupational therapy only. The patients also differed on all the variables collected. A short summary of these data is given below. (Further details can be found in Wykes *et al*, 1990.)

The majority had a poor record of work achievement, few had lived independently and many had contacted the psychiatric services many years previously. However, there were people who had had some skilled work and who had been married with some parental responsibility. There were 28 schizophrenic patients and 21 with other 'diagnoses'. Diagnoses were based on three sorts of information: CATEGO class (Wing & Sturt, 1978) derived from the PSE, syndrome check-lists on the case notes, and clinical diagnoses in the notes. Two of these three sorts of information had to match before a diagnosis of schizophrenia was given. This is a very strict criterion for schizophrenia and it might be argued that it is an underestimate of the level of the disorder in this sample. This strict diagnosis was not related to level of care received but chronicity of illness and level of job and living skills were. An expanded group of schizophrenics are correlated with other variables in this study to investigate the validity rather than the reliability of different criteria in the diagnosis of schizophrenia. Patients mainly showed non-specific neurotic symptoms. One-fifth had a delusion and one-third had some florid/positive symptoms. More than three-quarters showed negative symptoms. The various symptom measures were not related to the type of care in which patients were found. The level of problem behaviour was very similar to that collected on a community sample in Camberwell (Wykes *et al*, 1982). The median number of problems on the SBS (mild and severe problems score) in each of the three groups was (a) sheltered workshop, BSM = 3; (b) rehabilitation villa, BSM = 4; (c) long-stay ward, BSM = 3. No particular problem behaviour was related to level of care. However, the overall level of problems was related to level of care.

The cross-sectional study investigated four levels of difficulty in information processing. These were: a simple reaction-time (RT) task, two conditions where a load was placed on the stimulus-processing stages, and a further condition which tested response-processing difficulties. The loading was achieved by increasing the number of stimuli from a simple RT task with only one light and one response button to two lights and two buttons and finally, two conditions containing four lights and four buttons. In the stimulus-processing conditions the response was compatible, i.e. to turn off the light the subject pressed the button below it.

Response processing is gleaned in the following way. Many people have argued (e.g. Wickens, 1974; Hemsley, 1976) that when a response is compatible (i.e. natural) it allows the subject to bypass the response-processing stages. When the number of stimulus options are increased and the response is natural (i.e. the response button is paired to the light), there is only an increasing load on the stimulus-processing capabilities of the subject. In order to place a load on the response-processing stage, the subject is required to make an incompatible response. In this experiment the response was to turn off the stimulus light using the response button which was immediately clockwise to it. The difference between the RT for the simple task and that for the other three conditions provided measures of the stimulus- and response-processing difficulties of this group. There are several criticisms of the additive method of assessing information-processing stages. The main limitation is to assume that processing is serial rather than parallel and that the effects on different stages are independent. Despite these criticisms there is evidence that response-processing measures derived from Sternberg's method are related to the cognitive deficit in schizophrenia (Hemsley, 1976; Checosky in Wishner *et al*, 1983).

Figure 1 shows measures of stimulus and response processing for subjects in each of the three general care settings. The increase in RT when a processing load was placed on the stimulus-processing stage was minimal for the three groups. However, when a load was placed on the response-processing stage, there was a large increase in RT and this was particularly noticeable for patients in the long-stay ward. When the latter group were looked at in more detail it became apparent that there were two separate groupings of response-processing times. One group had a median of 517 ms whereas the other had a median of 1835 ms, with an intergroup interval of 433 ms. None of the above variables were related to this split in the sample (see Table I). In other words, both groups had high levels of negative symptoms and behaviour problems, and although a higher proportion of the patients had an experimental diagnosis of schizophrenia, this was not statistically significant.

It was proposed that the problems with response processing may be of direct relevance to the future potential of patients (Wykes *et al*, 1990). Difficulties in response

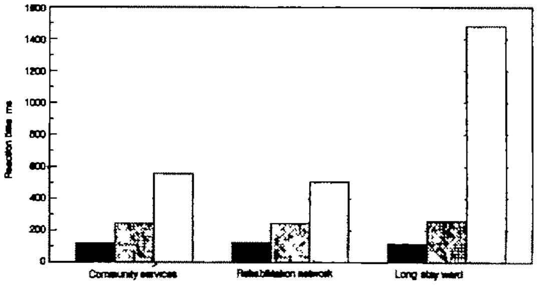

FIG. 1 Measures of stimulus processing (■ ▨) and response processing (☐) on a choice reaction-time task in the three general care settings (each measure shows difference between RT on a simple task and that on a task in which a load is placed on either stimulus or response processing).

TABLE I
The relationship between response processing and symptom and behaviour factors in the long-stay group (n = 23)

Factor	*Fast responders*	*Slow responders*
Average social behaviour problems[1]	4.2	4.5
Average positive symptoms[2]	1.9	1.25
Average negative symptoms[2]	2.1	2.75
Diagnosis of schizophrenia	53%	75%

1. Measured on the Social Behaviour Schedule.
2. Measured on the PSE.

processing may reflect a continuing underlying abnormality. Those patients who do not show problems with response processing but still show rather disabling symptom and behaviour patterns may constitute a group of patients whose rehabilitative potential is high and who may in the past have been labelled as the institutionalised patient. This latter group, as suggested by Hemsley (1977), may have adopted behaviour patterns as strategies to deal with cognitive overload, and continued to exhibit these behaviours when the cognitive abnormality disappeared. These behaviour patterns are then maladaptive and may be available to amelioration. Further details of these RT data can be found in Wykes *et al* (1990).

Procedure and analyses

The patients' use of all psychiatric services was noted from case notes, supplemented by discussions with staff. Three specific dates were chosen as markers. These were at six months, 18 months and three years after the initial data collection. Service use was divided into day and night care and these were further classified into nine and seven categories respectively as follows:

> *day care* (a) on the ward, no day care, (b) on the ward, ward helper, (c) occupational therapy, (d) industrial therapy, (e) structured tasks in a group home or hostel, (f) sheltered work, (g) employment rehabilitation scheme, (h) open employment, (i) none;
> *night care* (a) acute in-patient psychiatric ward, (b) long-stay ward, (c) supervised hospital villa, (d) unsupervised hospital group home, (e) staffed hostel, (f) group home or unstaffed home, (g) living in own accommodation.

None of the researchers had any influence on the placing of patients in the various psychiatric services. They also did not provide access to the initial data so staff who made decisions on the type of care needed were completely unaware of the results of the previous study. Staff were probably relying on their impressions of behavioural and symptom information but they were definitely not using the reaction-time data. Thus, there is no way that these data could have influenced placement.

Factors chosen from the initial study as possible determiners of rehabilitative success included: positive and negative symptoms, total number of symptoms on the PSE, level of social behaviour problems, reaction-time variables, chronicity, peak level of job skills, and peak level of living accommodation. They were entered into stepwise discriminant analyses using SPSS-X (1983) with day or night care as the dependent variables. Level of difficulty in response processing was also used independently to assess whether it was related to moving to more independent levels of care.

TABLE II
Number of patients who changed their care setting at each stage of the follow-up

Follow-up date	*No. who moved*[1]	
	Day care	*Night care*
6 months	11	9
18 months	20	11
3 years	21	12
Total who moved at any time during the 3 years	26	16

1. To more or less independent forms of care.

Results

Several people moved their day and/or night care during the three years of the follow-up. Many of these were improvements but there were also some increases in the dependence on psychiatric care. Table II shows the number of people who moved at each step in the follow-up period irrespective of the direction of their move (i.e. to more or less independent forms of care). This includes people who moved several times as well as those who moved to a different form of care and were then transferred back. During the follow-up one patient died and so is not included.

Discriminant analyses were carried out on the patients' care settings at each step of the follow-up and for day and night care separately using the variables from all the categories specified above. The reaction-time data were transformed to obtain some independence of the mean and variance, but one outlier was still excluded from all the parametric analyses. The correct classification rates based on the factorial structure devised by the program are shown in Table III.

In all the analyses, reaction-time variables were included in the predictor set. However, the predictor sets were different for each step of the follow-up. In general, there

TABLE III
Stepwise discriminant analyses (all diagnoses)

Follow-up date	*Correct classification rates* *day care*	 *night care*
6 months	70%	55%
18 months	60%	59%
3 years	64%	64%

was a lower correct classification rate for the schizophrenic group than for the group of mixed diagnoses. The predictor set for the schizophrenic group was also characterised mainly by reaction-time, social and demographic factors whereas the 'other diagnoses' group was more likely to have symptoms in the predictor set.

The amount of variance in service use accounted for by the response-processing measure was significant only for night care of schizophrenic patients (25% of variance at six-month follow-up, $P<0.009$; 25% at 18-month follow-up, $P<0.009$; 30% at three-year follow-up, $P<0.004$).

The definition of schizophrenia adopted in this study was based mainly on symptom descriptions by the patients themselves and those in the case notes. These descriptions may be a reliable way of assessing schizophrenia, but not the most valid. Shepherd (1987) suggested that cognitive dysfunction, once thought to be the fundamental difficulty for schizophrenics, has now achieved less prominence as researchers have concentrated on the assessment of delusions and hallucinations. If in this study we were to attempt to identify the optimal validity of a diagnosis of schizophrenia in terms of extra-class correlates (e.g. service use), we would perform a 'boot-straps operation' and include those patients who had slowed response-processing times from the 'other diagnoses' group in the schizophrenic sample. When this was done the response-processing measure accounted for less of the variance in service use, although the difference was minimal. Some or all of the group of patients who show slowed response processing but not the key schizophrenic symptoms rated on instruments such as the PSE may be those who would fall into a diagnostic class of simple schizophrenia. However, they could also be another separate class of diagnoses altogether. In order to clarify these possibilities further detailed analyses need to be carried out on other extra-class correlates such as response to phenothiazines.

The response-processing measure only seems to account for a significant amount of variance in night-care placements for the schizophrenic group. However, for this group it was highly significant. One explanation for this differential performance in accounting for day and night care could be the differences in the numbers of places available. It is imperative that patients be provided with a roof over their heads so that they all have a night-care setting. This placement is likely to be appropriate to their needs because there is always the relevant number of placements available which allows for some flexibility. Day care, unfortunately, does not have such a high priority, there are fewer places and so is less likely to be allocated in an appropriate fashion, as many patients cannot be provided with a day-care option.

TABLE IV
Relationship between response processing and moving at any time within three-year follow-up

	Moved within 3 years *day care*	 *night care*	*Moved to more independent care* *day care*	 *night care*
Slow responders	45%	9%	18%	0%
Fast responders	54%	38%	38%	35%

In the previous analyses, response processing was treated as a continuous variable, but we were particularly concerned with those patients who had difficulties with this processing stage. In the cross-sectional study a bimodal distribution of scores was found. We therefore divided the group into those patients who experienced difficulties (processing time > one second) and those who did not. The relationship between slow responses and moving to different forms of care at any time during the three years following the initial data collection period is shown in Table IV.

From Table IV it is obvious that people showing slow response times were less likely to move to more independent care during the three years they were followed up. The difference between the slow and fast responders in moving to more independent levels of care, however, was statistically significant only for night care (night care, $\chi^2=4.2$, $P<0.05$; day care, $\chi=0.7$, NS). The patients with slow response times did move (45% changed their day care) but they transferred to services that provided *more* psychiatric care not less. Although this table covers all diagnoses, the results are similar for the schizophrenic and 'other' diagnostic group. This same pattern can also be observed in the group of patients who were least likely to move – those in the long-stay ward. Thus, some patients did gravitate to less dependent care (day care, 29%; night care, 27%) but these were people who had previously shown few problems in the response-processing stage of the RT task. In fact, no patient showing a slow response-processing time moved to more independent levels of care during the three-year follow-up.

It was possible to discriminate between the movers and non-movers using five variables including chronicity, measures of previous employment skills and independent living, social behaviour problems, and slow response processing. It was also possible to correctly classify 77% of these two groups using only these five variables.

Discussion

This follow-up study has exploded one myth about chronic patients – that they remain in the same service setting for long periods of time. Given that there are services that can be allocated appropriately there can be a great deal of movement – not all of it to more dependent levels of care. Thus, even after a considerable period in a psychiatric hospital, there is still some flexibility of outcome.

The outcome over the three years could be predicted by the loading on the factors identified in the cross-sectional study. These factors included symptom, behavioural and demographic variables as well as the level of performance on a response-processing task. This latter measure alone was able to account for a considerable amount of variance in the night-care arrangements for the schizophrenic group at each step in the follow-up. When the sample was split into slow and fast responders, the slow responders were the group who did not move to more independent levels of care. The slow responders did not differ from the fast responders on symptoms, behaviour or demographic characteristics and were postulated to be still suffering from an underlying cognitive impairment. The fast group were thought to consist of people whose original cognitive impairment had disappeared but whose strategies adopted to deal with the cognitive impairment were still prevalent. In other words, this group were institutionalised and were therefore capable of participating in, and benefiting from, traditional rehabilitation programmes.

Although it is difficult to quantify the level of response processing required by different environments it seems obvious that there is a dimension of predictability built into rehabilitation environments. In general, a long-stay ward is more regular and predictable than life outside hospital. The number of options in the community will be wider and will therefore be affected by the ability to carry out response processing. The increased level of uncertainty may be disadvantageous for those who still show prominent cognitive abnormalities. If further advances into community settings are to be made for the patients showing these abnormalities, they may only be achieved if community settings are made more predictable.

Some positive results were found from this study which could be used in the short-term prediction of successful movement through the services, but there is also the need to continue following up such a group. Some people did change the type of care they were receiving and these patients were the ones who had fast response processing. However, it is not yet clear whether: (a) people who have slow response-processing times cannot ever take advantage of traditional rehabilitation practices (it is possible that when rehabilitation services are limited the patients chosen to be transferred initially are those with faster response-processing times); (b) the response-processing measure is stable over time or is related to mental state or current level of skills.

In order to test (a) another follow-up period is required during which further efforts should be made to transfer these patients to less dependent care. Netherne Hospital is now scheduled to close and services in the community will be expanded. Patients will be provided with extra support in order to transfer them into these new community services. This state of affairs will provide a stringent test of the usefulness of response processing in predicting outcome. The services will again be making decisions about patient care which will not be dependent on any of the initial measures from the cross-sectional study and will be independent from the research team. A further follow-up is therefore planned in three years' time.

Investigating whether response processing is stable over time would throw some light on the status of response processing as a measure of vulnerability. Neuchterlein & Dawson (1984) suggested that there are three levels of vulnerability factors. At the first level are the stable vulnerability factors which are trait-like characteristics that are consistently deviant from normal levels even during periods of remission. At the second level are mediating vulnerability factors which are deviant from normal levels during psychotic and asymptomatic states but also covary with level of symptoms. Finally there are the symptom vulnerability factors which index internal processes that are associated with certain psychiatric features. The previous cross-sectional study indicated that response-processing measures were not episode markers as they did not seem to be related to symptoms as measured on the PSE. However, in order to evaluate clearly whether this variable falls into either of the remaining categories, a longitudinal design is required because cross-sectional studies cannot rule out possible cohort differences. It is therefore necessary to employ a repeated-measures design. The planned follow-up study therefore will also include the collection of further reaction-time, symptom and behavioural data.

If response processing is a useful and/or stable indicator of lack of response to traditional rehabilitation methods then the group showing slowed response processing needs to be investigated in more detail in order to identify skills, processing or rehearsal strategies that might be utilised for successful rehabilitation. The metaphor adopted here is of special education where the skills of a handicapped group are developed by expanding further resources. We are not in favour of using variables, such as response-processing difficulties, to exclude certain groups from access to rehabilitation services, but to use the information on lack of effects to design more adaptive rehabilitation mechanisms. Shepherd (1978) has suggested that for social skills training "a slow rate of presentation, and some kind of pure part learning or progressive part learning may reduce other information-processing problems". The breaking down of daily living tasks into more manageable components which place only a small load on an individual's response-processing mechanism may be now achievable if we can use the performance on the response-processing task as a guide to the level of the breakdown required.

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Cognitive-Behavioural Problem Solving in the Treatment of Patients who Repeatedly Attempt Suicide

A Controlled Trial

PAUL M. SALKOVSKIS, CHRIS ATHA and DAVID STORER

In a controlled trial, 20 patients at high risk of repeated suicide attempts were randomly allocated to either cognitive-behavioural problem solving or a 'treatment-as-usual' control condition. The group practising problem solving improved significantly more than controls on ratings of depression, hopelessness, suicidal ideation and target problems at the end of treatment and at follow-up of up to one year, and there was evidence of an effect on the rates of repetition over the six months after treatment.

There is a need for "more research . . . to establish the most effective patterns of care for patients who deliberately harm themselves, while at the same time making better use of scarce resources" (Department of Health and Social Security, 1984). Methodological difficulties involved in carrying out such research have been well summarised by Hawton (1989). Self-harm occurs in a wide range of clinical conditions, and is not uncommon among those without any formal psychiatric diagnosis (Hawton & Catalan, 1987). This suggests that pharmacotherapy for repeated suicide attempters may be of questionable value, and that psychological treatment is more appropriate. Furthermore, there is evidence that drug treatments are ineffective (Morgan, 1979); moreover many patients attempt suicide using prescribed psychotropic medication. For a psychosocial intervention to be useful in self-harm patients, it must be brief, flexible, and relevant to a wide range of difficulties and diagnoses. Recent experimental studies of patients who have attempted suicide have shown specific deficits in problem-solving abilities (e.g. Linehan *et al*, 1987; Schotte & Clum, 1987), consistent with the hypothesis that attempted suicide may relate to failures of problem solving at times of crisis.

Taking all of these factors into account, the cognitive-behavioural approach to problem solving should be a useful short-term treatment, and it has met with some limited success when applied to the treatment of self-harm cases (Hawton, 1989). Treatment involves the therapist teaching patients to deal systematically with life stresses and then to apply the same principles on their own (Hawton & Kirk, 1989).

The four studies of psychosocial interventions thus far carried out with suicide attempters have failed to find specific benefits compared with control conditions (Chowdhury *et al*, 1973; Gibbons *et al*, 1978; Hawton *et al*, 1981, 1987). Some studies have found effects of treatment on social adjustment, or have found a treatment-by-sex interaction (Hawton, 1989). However, although the treatment effects have been limited, many of the difficulties encountered might be overcome by: (a) choosing a sample in which spontaneous remission of problems after the index attempt was relatively unlikely (i.e. a group of patients at high risk for repetition of suicide attempt); and (b) using a range of both standardised outcome measures, to evaluate general characteristics of the population being studied (e.g. levels of depression and hopelessness), *and* individualised outcome measures, to take account of the diagnostic heterogeneity of self-harm patients. A problem in evaluating the treatment of patients at high risk for repeated self-harm attempts is the difficulty in getting such patients to attend hospital appointments (Hawton, 1989).

The present study evaluates a cognitively-based problem-solving treatment in a population at high risk for repeated self-harm, delivered in the patients' own homes.

Method

The study was conducted at the Leeds General Infirmary (LGI), a large inner-city general hospital serving half of the city of Leeds. Subjects were recruited from patients seen by the duty psychiatrist following a reported suicide attempt. In order to be eligible for the study, patients had to be aged 16–65, living within the Leeds Western Health Authority boundary and of fixed abode, not judged by the assessing psychiatrist to require immediate psychiatric treatment, and not at present suffering from a psychotic or serious organic illness.

The main inclusion criteria had been determined from a previous (unpublished) study of predictors of repeated self-harm in the LGI. These were that (a) there had been two or more previous suicide attempts, (b) antidepressants had been taken as part of an overdose, and (c) patients scored at least 4 on the six-point scale devised by Buglass & Horton (1974) to predict repeated suicidal behaviour. (This scale is composed of the following items: previous diagnosis of sociopathy, problem in the use of alcohol, previous psychiatric in-patient care, previous psychiatric out-patient care, previous parasuicide admission, and not living with a relative). To be included, patients had to fulfil at least two of these three criteria.

Those patients fulfilling inclusion criteria were informed of the purpose of the study and asked to consent to participate. If informed consent was provided, patients were immediately seen by the research community psychiatric nurse (CPN), who followed a predetermined random allocation to experimental or control conditions. Twenty patients were included in the study, and all except four (two in each group) met the Buglass criteria. Twelve subjects were allocated to the treatment group, eight to the control condition (the imbalance in group sizes arose because the original design had called for *two* treatment conditions (see below – low referral rates prevented completion of the full design). Randomisation resulted in a slightly higher proportion of men in the control group; in all other respects measured here the groups did not differ in composition or characteristics. All patients consenting to admission to the study completed the assessments, and there were no treatment drop-outs.

All patients were medically treated in the accident and emergency (A&E) short-stay ward or other medical wards in the LGI. Before allocation, the assessing psychiatrist carried out a structured interview, during which he/she collected demographic data, took a psychiatric history, completed the Beck Suicidal Intent scale (Beck *et al*, 1974*a*) with respect to the actual index attempt, and determined whether the patient fulfilled the selection criteria. (Copies of the interviews schedule used as part of the routine overdose assessment at the LGI can be obtained from DS.)

All subsequent research assessments for all patients were conducted by the community psychiatric nurse (CPN) in the patient's own home, in order to maximise compliance. These assessments were at one week, one month, three months, six months, and one year after the index suicide attempt. The CPN completed the Beck Suicidal Ideation scale (Beck *et al*, 1979); this is a structured interview which measures thoughts, plans and attitudes related to *future* suicide attempts. Only scale I (Characteristics of attitude toward living/dying; e.g. 'Wish to live'', rated from 0, 'Moderate to strong', to 2, 'None') and scale II (Characteristics of suicide ideation/wish; e.g. "Control over suicidal action/acting out wish", rated from 0, "Has sense of control", to 2, "Has no sense of control") were analysed here, as the other scales all refer to a contemplated attempt, which was present in only a few subjects on the later assessments. At the first interview, the CPN and patient constructed a Personal Questionnaire using the Personal Questionnaire Rapid Scaling Technique (PQRST; Mulhall, 1977), in which the items are generated by the patient as a problem list. All of the patients main current or impending problems are ranked in order of importance. Although the problems are individually generated for every patient, the scaling of each problem is standardised. Furthermore, the use of paired comparisons allows a check of internal consistency to be made for the items each time they are scored. This measure is particularly useful with patients who have a wide range of difficulties. It is also particularly helpful in the context of cognitive-behavioural problem-solving approaches with patients experiencing psychosocial difficulties because it measures specific problems rather than psychiatric symptoms.

The number of problems identified varies between subjects. In this sample, up to three problems were identified and rated for all subjects as some had not identified more than three. In the present study, these problems were ranked by the patient in descending order of severity, so that problem 1 was the *most difficult*. At assessment, all patients also completed three self-rated scales: the Profile of Mood States (POMS; McNair & Lorr, 1964), a short scale giving subscale scores for depression, vigour, anxiety, tension, fatigue; the Beck Depression Inventory (BDI; Beck *et al*, 1961); and the Hopelessness scale (Beck *et al*, 1974*b*), which is of particular importance because of the strong relationship demonstrated between hopelessness scores and later suicide (Beck *et al*, 1985).

After initial assessment, the records of all patients admitted to the study were 'flagged' in the A&E records to allow collection of data with respect to repeat attempts.

Treatment

At initial assessment, the duty psychiatrist decided whether the patient required and would benefit from the range of treatment options normally available. If further treatment would not normally be offered, and patients were suitable in other respects, they were referred to the research project. This meant that the assessing psychiatrist discharged the patients with a letter to their general practitioner, which was the normal procedure. Potential CPN involvement as part of the trial was not a factor influencing treatment.

'Treatment as usual' was used as the comparison group because of the difficulties inherent in providing a credible control treatment (Teasdale *et al* (1984) give a fuller discussion of the utility of treatment as usual as a control condition). Our interest lay in establishing the clinical impact of the problem-solving treatment rather than in a component analysis of this therapy.

In the experimental group, patients were provided with additional treatment delivered by the CPN. In four cases this was entirely conducted in the patients' own home, while the remainder began treatment as in-patients during a 24-hour crisis intervention admission, and treatment was subsequently completed at home. Allocation to in-patient/home-based treatment was random; originally, the intention was to compare different modes of intervention, but the final sample size did not permit this. This also accounts for the uneven group sizes.

Treatment was based on a problem-solving approach (Bancroft, 1986; Hawton & Kirk, 1989); although a manual was not used, the format specified by Bancroft (1986) was used to provide guidelines which could be applied flexibly. Five sessions of treatment of at least one hour each were given by the CPN, at the time of the index attempt, three days later, a week after the attempt, two weeks after the attempt, and finally at one month after the index attempt (thus two treatment sessions coincided with assessment sessions). Homework assignments were used as necessary. Patients were taught how to identify problems and arrange priorities for problem solving. The next stage was to teach patients how to generate a wide range of solutions (by 'brainstorming') and narrowing this down to concrete and attainable goals which would represent a personally significant improvement. Next, strategies necessary to work out and implement steps towards realising these goals were considered, together with ways of determining and monitoring success. Emphasis was also placed on the importance of being flexible on the basis of results obtained, then deciding new or further goals as necessary (see Hawton & Kirk (1989) for a detailed account of problem-solving procedures).

The therapist (CA) was an experienced CPN with extensive experience of working with suicide attempters (see Atha *et al*, 1989; Salkovskis *et al*, 1989).

Statistical analysis

Results were analysed using a repeated-measures analysis of covariance (ANCOVA), with treatment as a grouping factor. Scores on each measure at the first assessment were used as covariate, with assessments at one week, one month, three months, six months and one year as dependent variables. The most important effect for this comparison was, therefore, the overall group effect; where such an effect is significant, it would indicate an overall difference between experimental and control groups. A group by repeated measures (time) effect would indicate different rates of change in the two groups; this was considered an unlikely outcome, given the brevity of treatment. Wherever the effect being considered involved repeated measures (e.g. a group by time interaction), a Greenhouse-geisser correction was applied. The program used for analysis was BMDP:P2V (Dixon *et al*, 1983).

TABLE I
Characteristics of the two groups

	Experimental group (n = 12)	*Control group (n = 8)*
Mean age (s.d.): years	26.4 (6.0)	28.5 (7.9)
Male: female sex ratio (% female)	5:7 (58%)	5:3 (38%)
Marital status		
single	6	2
married	3	3
separated	1	0
divorced	2	3
Previous psychiatric study		
in-patient (%)	4 (33%)	3 (37%)
out-patient (%)	7 (58%)	5 (62%)
Mean no. of previous attempts (s.d.)	2.6 (0.9)	3.0 (0.9)
Mean score on Beck suicidal intent scale (s.d.)		
objective circumstances	4.4 (2.0)	3.6 (1.6)
self-report	6.8 (1.9)	7.4 (2.3)
Mean score on Buglass risk of repetition scale (s.d.)	3.7 (0.8)	3.5 (0.9)

Results

The patient characteristics of the two study groups are shown in Table I. Regarding the Beck Suicidal Ideation scale, for scale I there was no effect of group ($F(1,17) = 1.59$, $P > 0.05$), and no group by time effect ($F(4,71) < 1$) (Fig. 1(a)). For scale II, there was a significant group effect ($F(1,17) = 6.08$, $P < 0.025$), but no group by time effect ($F(4,72) < 1$) (Fig. 1(b)).

On the PQRST, there was a significant group effect for problem 1 ($F(1,17) = 11.52$, $P < 0.005$), but the group by time effect was not significant ($F(4,72) = 1.03$, $P > 0.05$) (Fig. 2(a)). For problem two, the group effect was also significant ($F(1,17) = 15.84$, $P = 0.001$), and the group by time effect was not significant ($F(4,72) = 1.28$, $P > 0.01$) (Fig. 2(b)). The third rated problem showed a significant group effect ($F(1,17) = 15.00$, $P < 0.005$), while the group by time effect was not significant ($F(4,72) < 1$) (Fig. 2(c)). Thus, the problem-solving group showed significantly better overall results on their three main problems when compared with the group who received 'treatment as usual'.

The initial BDI scores for experimental and control groups indicate high initial levels of depression (31.75 and 30.37 respectively). The ANCOVA results show a main effect of group ($F(1,17) = 11.86$, $P < 0.005$) and no group by time interaction ($F(4,72) < 1$). The means are shown in Fig. 3.

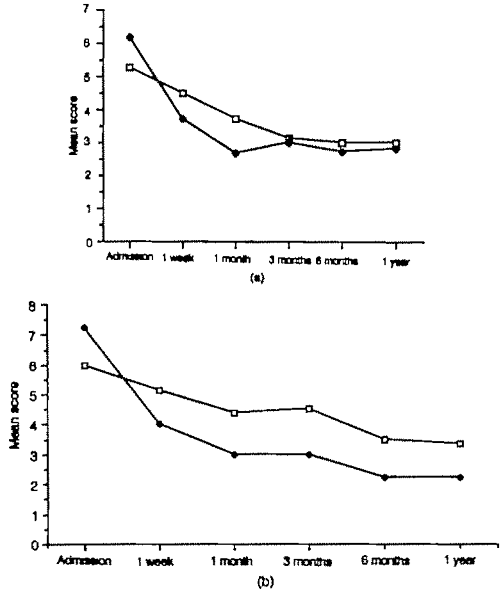

FIG 1 Mean scores on the Beck Suicidal ideation scales (□—□ controls, ◆—◆ treated group). (a) Scale I, attitude towards living/dying, and (b) Scale II, characteristics of suicide ideation/wish.

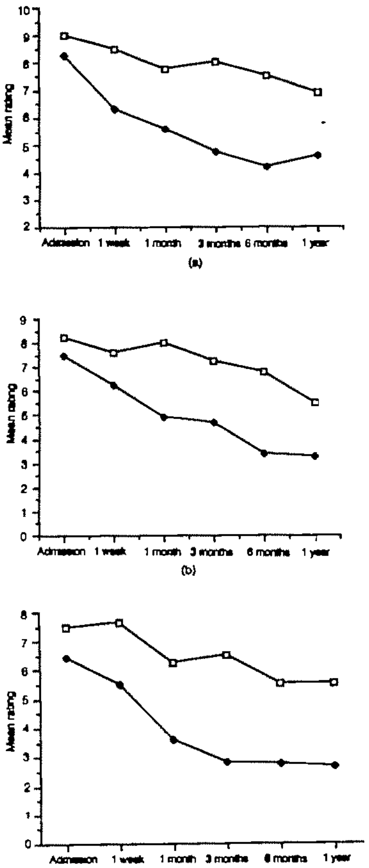

FIG. 2 Mean PQRST rating of severity of three main problems nominated by subject (a–c in order of increasing severity) (□——□ controls, ◆——◆ treated group).

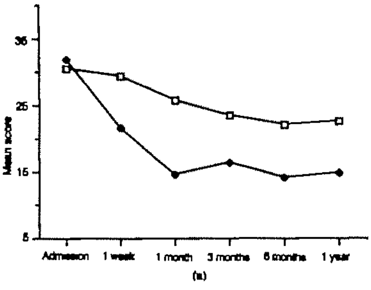

FIG. 3 Mean scores on the Beck Depression Inventory (□——□ controls, ◆——◆ treated group).

On the Hopelessness scale (Fig. 4), ANCOVA shows a significant main effect of group on post-treatment hopelessness scores ($F(1,17)=8.85, P<0.01$), with group by time effects not being significant ($F(4,72)<1$).

For POMS subscales, significant group effects were found for depression ($F=16.6, P<0.001$) and fatigue scales

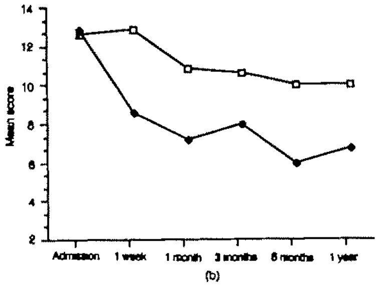

FIG. 4 Mean scores on the Hopelessness scale (□——□ controls, ◆——◆ treated group).

($F=7.33, P<0.05$) and a significant group by time interaction was found for tension ($F=3.76, P<0.01$).

Using Fisher's exact test, the difference between proportions of patients repeating a suicide attempt in the six months after the index attempt was significant (none in the experimental group v. three patients and four attempts in the control group; $P=0.049$). By 18 months, the figures are no longer significant (three patients and the three attempts in the experimental group v. four patients and six attempts in the control group; $P=0.19$). In the experimental (treated) group, the mean time to repeat was 9.3 months, while the mean time to repeated attempt for the subjects in the control group was three months. This descriptive data is not, of course, open to statistical analysis, but inspection suggests that repeat suicidal behaviour tended to occur later in the experimental group.

Post-hoc tests

Given the absence of significant group by time interactions, Tukey tests were carried out to examine whether treatment effects were already significant at the end of week one after two sessions of treatment and after one month. Covariance-adjusted means were used in these planned comparisons. Only the Hopelessness scale showed a significant difference between groups at week 1. The other main variables compared showed that the one-month differences were significant. Thus, for most variables, the significant differences emerge only on the completion of treatment.

Analysis of follow-up data

As treatment was concentrated in the period immediately after referral, it is possible that the group effect on the ANCOVA was unduly influenced by the first few assessments. In order to check this possibility, a further analysis was carried out, using only the six-month and one-year follow-up points; the first assessment was again used as covariate. The results of this analysis are similar to those in the full analysis. There were no significant time effects or time by group interactions. There was a significant group effect for BDI scores ($F(1,17)=8.1, P<0.05$), for the Hopelessness scale ($F(1,17)=9.2, P<0.01$), for the main problem, ($F(1,17)=13.14, P<0.005$), for problem 2 ($F(1,17)=9.62, P<0.01$), and for problem 3 ($F(1,17)=$

21.3, $P<0.0005$). There was no effect for Beck Suicidal Ideation scale I ($F(1,17)<1$); on the present analysis, scale II shows a non-significant trend on the group effect ($F(1,17)=3.4$, $P=0.07$).

Discussion

The results of this study suggest that a brief problem-solving treatment may be effective in reducing the distress experienced by patients at high risk for repeated suicide attempts. Short-term effectiveness in terms of repeated attempts was also demonstrated. The effectiveness of the treatment as indexed by measures of psychopathology was maintained during follow-up. The high initial level of psychopathology and distress (as indexed by depression and hopelessness scores) and high problem ratings are consistent with the selection criteria applied. Indeed, the characteristics of this group of patients (e.g. with chronic psychiatric, personality and alcohol problems) are such that many clinicians would regard treatment as impractical and likely to be refused and/or unhelpful to the patient. By definition, patients included in the present study were regarded by the assessing psychiatrist as not requiring (or not likely to benefit from) available treatments. Many such patients do not attend when offered a follow-up psychiatric out-patient appointment.

The use of an individualised outcome measure is an important advance in this type of study, in that it allows some indication of the impact of treatment on the main problems identified by each patient in this extremely heterogeneous population. This is especially relevant because treatment focused on training in problem solving: therapy was directed at teaching patients to learn problem-solving *techniques* rather than to the solution of specific individual problems. In most patients this meant that only their main problem was specifically targeted by the therapist; in none of the patients was the third ranked problem dealt with during therapy. Significant treatment effects were, however, found for all problems rather than just the main one, suggesting that patients had generalised the skills. A further important consideration was that no patients dropped out of treatment.

There are a number of problems in interpreting the results. The sample size is relatively small. The experimental treatment was in one of two modes (in-patient followed by home-based or just home-based); the small final sample size meant that we were unable to carry out the analysis of this factor, as originally intended. As is always the case in the evaluation of a treatment package of this kind, the relative contribution of different components of treatment is difficult to assess. Treatment was carried out by one therapist, and we cannot rule out the possibility that the treatment response was therapist-specific. In this study, audiotapes of therapy sessions were not used; this would have been helpful to ensure the integrity of treatment. The possibility that the results may have been due to non-specific factors such as different amounts of contact time is not consistent with the results of the study carried out by Hawton *et al* (1987). It is, however, quite likely that the repeated home-based assessments may have had a positive impact on the control group.

A further problem with a study of this size are possible pre-treatment group differences. There was a tendency for there to be more women in the treated group, and although sex did not emerge as a main effect in the analysis of covariance, the cell sizes are too small to allow proper interpretation of the absence of a group by sex interaction, so this cannot be totally ruled out. Clearly, replication is required in any case to ensure that there was not a failure of randomisation on some variable that was not measured.

The benefits of treatment persisted beyond the treatment period itself. The absence of a group by time interaction was probably because the bulk of treatment was concentrated in the first week; the rapid response in terms of the Hopelessness scale is almost certainly a reflection of the immediate impact of the first few sessions of the type of treatment used here. Previous studies have demonstrated only limited effects of psychological treatment, although they were able to tentatively identify subgroups of 'responders' (Chowdhury *et al*, 1973; Gibbons *et al*, 1978; Hawton *et al*, 1981, 1987; Hawton, 1989). One possible explanation of the relatively negative outcome of previous studies may thus lie in their populations. These have usually included a substantial proportion of patients after their first self-harm episode; only the study in Chowdhury *et al* (1973) used 'repeaters', and this study explicitly excluded those at particularly high risk. The selection of a more chronic and severe sample, as in this study, prevents treatment effects being masked by the substantial spontaneous improvements likely in the majority of patients after the passing of the crisis which precipitated the index suicide attempt. In other words, the patients must be selected in a way which ensures the untreated patients do not improve so much as to make it impossible to show any additional effect of treatment. Thus, previous studies have shown substantial improvements in both treatment and control groups (e.g. Hawton *et al*, 1981), leading to a possible floor effect.

It is particularly encouraging that treatment was associated with a short-term reduction in the frequency of repeat suicide attempts. The absence of a significant effect over the longer period is a problem; it is possible that a larger sample size might reveal a difference, or it may be that a longer-term active follow-up is required to sustain improvement. Another indication of the impact of treatment on patients is the *total* number of attempts (which could not be statistically analysed) and the significant reductions in scale II of the Beck Suicidal Ideation interview; there was also a substantial, significant and enduring reduction in depression and hopelessness scores in the experimental group compared with the controls (cf. Beck *et al*, 1985).

While by no means conclusive, the present study appears to indicate that a brief problem-solving treatment is effective in reducing the distress experienced by a group of repeated suicide attempters and has an impact on the rate of repeated suicide attempts in the short term. Consistent with previous results (Hawton *et al*, 1981), it seems likely that the delivery of treatment in the patients' own homes may have ensured good compliance.

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Patient Psychopathology and Expressed Emotion in Schizophrenia

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The relationship of a full range of psychiatric symptoms to EE was examined in 40 men with BPRS and SANS diagnoses of schizophrenia or schizoaffective disorder. Patients from high-EE families had significantly higher ratings of positive symptoms, anxious depression, and overall psychopathology, but not negative symptoms, than did those from low-EE families. In predicting relapses of schizophrenia, account may need to be taken of an interaction between subtle differences in symptoms and relatives' attitudes.

A constellation of relatives' critical comments and/or emotional overinvolvement, called 'expressed emotion' (EE), is associated with increased rates of relapse among recently discharged schizophrenic patients (Brown *et al*, 1972; Vaughn & Leff, 1976*a*; Vaughn *et al*, 1984). However, the relation between patients' symptoms and relatives' EE is unclear. In an early study, Brown *et al* (1972) found a strong association between patients' long-term work impairment and behavioural disturbance and their families' EE, but found little relation between patients' acute symptoms and EE. Mintz *et al* (1987) reanalysed data reported by Macmillan *et al* (1986) and found that patients from high-EE families exhibited more 'disturbed behaviour', but specific measures of psychiatric symptoms were not included. Vaughn & Leff (1976*a*) found a non-significant relationship between overall severity of patients' behavioural disturbance, as reported by their relatives, and EE. Unfortunately, these studies provided little information about the operational definitions and instruments used to measure patient symptoms.

In a study using more widely accepted measures of psychopathology, Miklowitz *et al* (1983) found no significant differences in schizophrenic patients' symptoms measured on either a pre-admission Present State Examination (PSE; Wing *et al*, 1974) or discharge Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) as a function of relatives' EE. Vaughn *et al* (1984) similarly reported no differences in family EE related to initial total PSE psychopathology, or total schizophrenic symptoms measured on the Psychiatric Assessment Scale (PAS; Krawiecka *et al* 1977).

Two reasons why previous research has not identified symptom differences between patients living with high-EE and low-EE relatives may be: (a) an underemphasis on measuring differences in *severity* of psychopathology rather than just their presence and absence; and (b) failure to assess multiple dimensions of patient psychopathology during or proximal to a period of symptom exacerbation. For example, Strachan *et al* (1986) have published data indicating worse depression in patients with high-EE families. Kanter *et al* (1987) have speculated on the potential relevance of negative symptoms to EE. Hooley (1985) suggested that high EE may reflect relatives' attempts to exert control over essentially uncontrollable behaviour. Within this framework, those symptoms that have the appearance of being more volitional (e.g. apathy, withdrawal) elicit more criticism than those that seem less under the patient's control (e.g. hallucinations, delusions). Thus, Hooley hypothesised that high EE, particularly the number of critical comments, would be associated with higher levels of negative symptoms. Koenigsberg & Handley (1986) postulated an alternative relationship between negative symptoms and EE. They propose that low-EE relatives may exhibit cool, detached demeanors which "quell the emergence of positive symptoms at the price of engendering withdrawal and apathy" (p. 1371). According to this hypothesis, negative symptoms would be more prevalent in low-EE families. These alternative hypotheses have not been tested.

We investigated the relations between dimensions of schizophrenic patients' symptoms and families' EE during or soon after a symptom exacerbation. Our goals were to test the competing hypotheses of Hooley (1985) and Koenigsberg & Handley (1986) regarding negative symptoms, as well as to identify other psychopathology discriminating patients from high-EE and low-EE families.

Method

Forty male veterans from the armed forces with a diagnosis of schizophrenia or schizoaffective disorder, featuring a

TABLE I
Mean patient psychopathology scores grouped by relatives' EE status

	Low EE	*High EE*	t *(d.f. = 38)*
BPRS subscale scores[1]*: mean (s.d.)*			
Schizophrenic thought	7.8 (4.9)	10.5 (3.5)	2.08*
Emotional withdrawal/ retardation	6.0 (2.8)	7.0 (3.3)	0.96
Hostile suspiciousness	5.9 (3.5)	6.7 (2.2)	0.81
Anxious depression	5.9 (2.1)	8.0 (3.3)	2.19*
Total	35.3 (13.0)	43.1 (10.1)	2.10*
SANS global items[1]*: mean (s.d.)*			
Affective flattening	1.7 (1.1)	2.0 (1.2)	0.53
Alogia	1.2 (1.2)	1.3 (1.4)	0.21
Avolition/apathy	1.9 (1.2)	1.9 (1.2)	0.01
Anhedonia/asociality	2.1 (1.3)	2.6 (0.7)	1.58
Attentional impairment	0.7 (1.0)	1.3 (1.3)	1.40

1. Possible ranges: 3–21 for BPRS subscales; 18–126 for total BPRS score; and 0–5 for all SANS items
*$P<0.05$

current or very recent symptom exacerbation, and their families served as the subjects. All diagnoses were made using DSM-III criteria (American Psychiatric Association, 1980) after PSE interviews and thorough reviews of medical records. All participants were part of a larger study to assess the efficacy of behavioural family management (Falloon *et al*, 1984). The data reported here were collected in the six to eight weeks before the intervention. Subjects were included if they were male, between the ages of 18 and 55, and spent (or were willing to spend) at least four hours each week with nearby family members during the family therapy. Subjects were excluded if they were transient, had a severe medical illness, organic mental disorder or seizure disorder, were dependent on alcohol or illicit drugs, did not speak English as their primary language, or had a medical history contraindicating the use of neuroleptics. All participants gave informed consent.

Patients' positive symptoms, as well as anxious depression and hostile suspiciousness, were assessed using subscale scores from the BPRS (Overall, 1976). Negative symptoms were specifically measured using the global items from the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1979) and the emotional withdrawal/retardation subscale from the BPRS.

All patient assessments were conducted during or subsequent to a clear exacerbation of psychotic symptoms documented by the treatment staff. Twenty-six patients were admitted to the hospital for this exacerbation. The remaining patients could be managed on an out-patient basis. Following a clear diagnosis confirmed using the PSE, all subjects were administered the BPRS and the SANS by one of six study psychiatrists trained to at least 80% inter-rater reliability and blind to family EE level. Random checks revealed the inter-rater reliability coefficient remained above 0.88 for all assessors throughout the study. Each rater completed the Global Assessment Scale (GAS; Endicott *et al*, 1976), an overall measure of patient functioning, after the diagnostic interview.

A member of the project staff contacted the relative(s) of each subject and invited them to participate in the project. They were asked to come in to sign formal consent and to be administered a battery of assessments including the abbreviated Camberwell Family Interview (CFI; Vaughn & Leff, 1976*b*) which is designed to measure EE (Leff & Vaughn, 1985). Briefly, it is a semistructured, tape-recorded 90-minute interview conducted individually with the relative which focuses on eliciting the respondent's description of and attitude towards the patient in the three months before hospital admission. The tape-recorded interviews were scored by one of two highly experienced raters who had been trained to reliability (greater 0.80 reliability coefficient with a criterion sample) previously. Tapes were rated for number of critical comments and degree of emotional overinvolvement. CFI scores of six or more criticisms and/or ratings of three (moderately high) or higher on emotional overinvolvement were designated as high in EE, in accordance with the criteria used by Vaughn *et al* (1984). Relatives not meeting these criteria were classified as low in EE.

In order to examine the relation between EE and symptoms, *t*-tests comparing the ratings of patients from high-EE and low-EE families on each symptom measure were conducted. Individual items on the BPRS were summed to yield four subscale scores (schizophrenic thought, anxious depression, hostile suspiciousness, and emotional withdrawal/retardation) and a total score. The SANS includes ratings on five global items (affective flattening, alogia, avolition/apathy, anhedonia/asociality, and attentional impairment), which were the major measures of negative symptoms used for analysis (see Table I).

Results

The mean age of the 40 patients was 30.5 (s.d. 5.7) years, and they had had a mean of 12.7 (s.d. 1.4) years of formal education. They had, on average, first been treated for psychiatric illness at the age of 21.4 (s.d. 4.9) years, and had been admitted for psychiatric problems a mean of 4.5 (s.d. 2.8) times. Eleven were white, 19 were black, eight were Hispanic, and two were Asian. Twenty-nine were unmarried, six were currently married, four were separated or divorced, and one was widowed. Thirty were unemployed. No differences in the relatives' level of EE were found on any of these patient variables.

The 40 relatives had the following kin relations with patients: five fathers, 20 mothers, seven spouses, six siblings, and one uncle. In the four dual-parent families, both relatives participated, but as the parents had similar EE levels, the mother's data only were examined in the statistical analyses. In all married couples, the spouse was the key family member. Thirty patients had lived with their participating relative before the symptom exacerbation;

39 had had at least four hours of face-to-face contact per week in the last month. Twenty-six of the 40 relatives were found to be high EE (14 on critical comments alone, 8 on emotional overinvolvement alone, 4 on both).

Patients from high-EE families exhibited more positive symptoms (Table I) as measured on the schizophrenic thought subscale of the BPRS. In examining the specific items which comprise the subscale, patients from high-EE families displayed significantly more unusual thought content (delusions) ($t(38)=2.66$, $P<0.02$), but not more hallucinatory behaviour ($t(21)=1.25$) or conceptual disorganisation ($t(38)=0.94$).

There were no significant differences associated with level of relatives' EE on any global items of negative symptoms. Similarly, there were no significant differences associated with EE on the BPRS emotional withdrawal/psychomotor retardation subscale. Thus, there is no support for the hypothesis that patients from high-EE and low-EE families differ in their level of negative symptoms during a period of exacerbation.

Patients from high-EE families exhibited significantly more anxious depression on the BPRS. Of the three items which comprise this subscale, patients from high-EE families were significantly more depressed ($t(38)=2.12$, $P<0.04$) and marginally more anxious ($t(38)=1.74$, $P<0.09$), but did not feel more guilt ($t(38)=1.2$).

Patients from high EE families exhibited a greater overall level of BPRS symptoms, as well ($t(38)=2.1$, $P<0.05$). No differences were obtained on any other BPRS subscale. Overall level of functioning, as measured by the GAS, did not differ between the two groups ($t(29)=0.81$).

The number of critical comments was significantly related to scores for BPRS schizophrenic thought ($r=0.28$, $P<0.05$, one-tailed) and the anxious depression subscale ($r=0.31$, $P<0.05$, one-tailed). Frequency of critical comments was associated with the SANS global item measuring anhedonia/asociality ($r=0.38$, $P<0.01$, one-tailed), but was unrelated to all other global SANS items.

Discussion

Patients from high-EE families had more positive symptoms and anxious depression than those from low-EE families. The number of critical comments expressed by relatives was associated with the patient's level of schizophrenic thought disorder (particularly delusions), anxious depression (particularly depression), and anhedonia/asociality. There was no support for any hypothesised relation between EE and negative symptoms.

The prominence of positive and depressive symptoms here is in contrast to data presented by both Vaughn *et al* (1984) and Miklowitz *et al* (1983), who report no differences in symptoms between patients from high-EE and low-EE families. However, neither group of investigators presented data on the *severity* of symptoms, but rather on patients' initial PSE ratings. Thus, while the PSE provides general information on psychopathology, the BPRS is a more appropriate instrument to measure more subtle differences in the severity of specific symptoms among a homogeneous group such as relatively young male adult patients with schizophrenia. Vaughn *et al* (1984) also reported that the total PAS score, a measure of symptom severity, was not related to EE. In our sample, however, patients from high EE families did evidence greater overall level of symptoms.

While many authors have postulated that the withdrawal and apathy associated with negative symptoms are especially aversive to families, in our data only schizophrenic thought, anxious depression, and overall BPRS symptom level were associated with EE. It may be, as Mintz *et al* (1989) found, that a greater proportion of patients from high-EE families here were non-compliant with outpatient treatment before this episode. This lack of treatment might have resulted in the development of symptoms which might have created more family tension and higher EE. Unfortunately, we do not have the data to assess this.

Alternatively, it may be that at least some negative symptoms, as measured in the clinic, have relatively little correspondence with behaviour in non-clinical settings, and thus little relation to family attitudes. Ratings on the SANS are generally based on observation and responses during an interview. Some negative symptoms, such as inattention and alogia, may be accurately observed during an interview. Other traits, such as avolition and anhedonia, are based primarily on patient self-report, but studies validating these reports are lacking (Lewine & Sommers, 1985). Since it is these latter traits that have been hypothesised to trouble families, a problem with the instrument as typically used may have precluded finding a strong relation with family attitudes here.

The validity of the negative symptoms assessment does have some support here, however. Number of critical comments was associated with the global SANS anhedonia/asociality item, which is rated on reports of hobbies, family intimacy, sexual interests, and number of friends. This finding is in accord with results of relatives' surveys (e.g. McElroy, 1987), which highlight patients' social deficits as a particular problem for relatives. This association suggests family complaints may have some basis.

Our finding that the severity of initial symptoms varies with EE sheds little light on the conundrum of causality – whether the stress of living with a high-EE relative produces more patient symptoms, or whether higher levels of patient symptoms result in higher levels of family EE. In fact, a one-directional causality is likely much too limited. A more complex interactional model, incorporating families' overall behavioural style, and specific responses to stress, as well as patient characteristics, is probably more accurate. In many families, distressing symptoms may elicit critical remarks, and critical remarks may result in a worsening of symptoms and contribute to relapse. Moreover, in some families, distressing symptoms may be more likely to elicit critical remarks, and patients may be more sensitive to them. The interactions among variables envisioned here are of critical importance and merit further study. More investigations of the *transactions* between schizophrenic patients and their families, as outlined by Strachan *et al* (1989), are clearly warranted.

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Somatosensory Evoked Potentials in Schizophrenia

A Lateralisation Study

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The SSEPs obtained from 19 schizophrenics defined by RDC, DSM-III and PSE criteria were compared with those from a control group of healthy volunteers. Previous findings of an abnormal lack of lateralising response in schizophrenic patients were not replicated. No significant difference in either amplitude or morphology between the traces obtained from the two groups were recorded. Ipsilateral and contralateral latencies for stimulation of the left and right index finger showed no significant difference in peak latency for any component between patient and control group. When mean peak-to-peak amplitudes were plotted the contralateral component was always greater in amplitude than the ipsilateral one. An objective measure of the degree of lateralisation, the percentage lateralisation quotient, showed no lateralisation differences between the patient and control groups. A case of myogenic contamination of ipsilateral components was observed calling into doubt findings where no temporal region monitoring has been performed.

In 1970, Hardin & Castelucci hypothesised that the latency difference between selected components of the ipsilateral and contralateral somatosensory evoked potential (SSEP) could be interpreted as representing commisural delay. Salamy (1978) performed SSEP recordings over a wide age range, employing a vibrotactile stimulus and recording electrodes over both somatosensory 'hand areas' referenced to the vertex (Cz) electrode, and observed an ipsilateral response of longer latency and lower amplitude than the contralateral response. This difference in latency decreased with age and was said to reflect maturation in somatosensory commisural transmission.

The origin of the ipsilateral somatosensory evoked response remains open to question. Although it may arise from an afferent volley originating from the contralateral cortex mediated via an interhemispheric pathway (the corpus callosum), the potentials could equally result from volume conduction from contralateral cortex, or by direct uncrossed pathways arising in the thalamus. Rosenthal & Biggelow (1972) showed a 20% thickening of the corpus callosum in a post-mortem study of schizophrenic subjects.

Jones & Miller (1981), inspired by the previous studies, set out to investigate the relationship between ipsilateral and contralateral SSEP components in schizophrenics. Using a recording protocol similar to that employed by Salamy, they found that the ipsilateral response in controls was small in amplitude and delayed in onset compared with the contralateral response. In schizophrenics, however, no delay was seen on the ipsilateral hemisphere, and it was suggested that ipsilateral pathways originated in the brainstem, rather than in a non-conducting corpus callosum. It was further proposed that schizophrenia might arise from agenesis of the corpus callosum.

In criticism of this work, Connolly (1982) observed that the technique employed would only assess the small number of myelinated callosal axons. Generalisation from this small group to conclusions of total callalosal block were felt to be ill-advised. However, in view of the potential importance of these findings, Shagass *et al* (1983) carried out a replication study after certain methodological faults had been removed. In Jones & Miller's original study, SSEPs had been recorded with a vertex (Cz) electrode used as reference. This electrode can be active, and a spurious ipsilateral response could therefore be recorded (Desmedt & Brunko, 1980). For this reason Shagass *et al* (1983) employed linked ears as well as a vertex reference site. Their findings showed contralaterally higher amplitude SSEPs in both controls and schizophrenics. They found no difference in the latencies between the ipsilateral and contralateral SSEPs in either group.

These and further conflicting studies (Tress *et al*, 1983; Fenwick *et al*, 1983) encouraged Cooper *et al* (1985) to investigate the matter more. SSEPs arising from a complex vibrotactile stimulus to the fore-fingers, similar to the stimulus employed by Jones & Miller (1981), were further studied. The stimulus, mainly of movement and to a lesser extent of touch, was modified to give an optimal tracing. Eight patients with affective disorder (three depressive and five manic) all had the expected contralateral hemisphere lateralisation as did 12 out of 15 normals. Three normals and three schizophrenics had a loss of lateralisation of the evoked response to stimulation of one hand and normal lateralisation when the other hand was stimulated (unilateral abnormality). Ten out of 21 schizophrenic patients had an abnormal lack of lateralisation response whichever hand was stimulated (bilateral abnormality). The wave forms were characterised by major positive peaks with a latency of approximately 30 ms and 45 ms. In control groups, the response recorded ipsilaterally was invariably too small to identify peaks reliably. Judgements on the degree of lateralisation were made subjectively by visual inspection. Lateralisation characteristics were evident only for the earliest part of the responses and at longer latencies there were often similarities in amplitude between the wave forms recorded over the two hemispheres.

It was claimed that the results could not be compared directly with those elicited by the mainly vibrotactile stimulus of Shagass *et al* (1983), or indeed, with those elicited by the commonly used artificial electrical stimulation of the median nerve (Fenwick *et al*, 1983). A further study that might replicate these findings was urged by the authors.

The following study was carried out using digital electrical stimulation, with the results recorded on the Biologic Brain Atlas III system to facilitate more detailed topographical analysis of the data.

Method

A group of 19 schizophrenics, both in-patients and out-patients, were compared with a group of normal healthy volunteers. Patients were interviewed using a modified form of the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1975) and included in the study if they fulfilled Research Diagnostic Criteria (RDC; Spitzer *et al*, 1975) and DSM-III (American Psychiatric Association, 1980) criteria for schizophrenia. Basic demographic data and clinical details, such as the duration of illness, were collected. A case-note review, using the Present State Examination (PSE) symptom check-list (Wing *et al*, 1974), allowed all patients to be classified as having nuclear schizophrenia.

Twenty electrodes were placed over the scalp according to the international 10–20 system at points Fp1,Fp2,F3,F4, F7,F8,Fz,Cz,Pz,T3,T4,T5,T6,C3,C4,P3,P4,O1,Oz and O2 with linked earlobe electrodes as the reference. The stimulus was produced by ring electrodes placed on the distal phalanx of the index finger, with cathode proximal to the anode. An earth lead held by a velcro strip was attached on the forearm; some light abrasion of the skin with the velcro beforehand produced a low resistance. Subjects were placed in a prone position, made warm and comfortable and asked to remain still with their eyes closed but to remain awake. Subjective threshold of the stimulus was ascertained and a current level three times this threshold used to elicit the SSEP. Stimulus duration was 200 μs and current levels were typically 3–6 mA.

Both right and left index fingers were stimulated in each subject, the order being on a random basis between subjects. Stimulation rate was 2.7 per second and 1024 stimulus runs were averaged. Careful monitoring of the input signal with automatic artefact rejection was maintained to ensure artefact-free recordings.

The Biologic Brain Atlas III system with built-in amplifiers was employed for recording. Gains were set at 50 000 with low-frequency filters at 10 Hz and high-frequency filters at 1500 Hz (-3dB points, roll-offs 12 dB octave). Twenty channels of data were recorded and stored on a 10-megabyte disc cartridge for analysis. The system allows generation of data in the form of isopotential colour contour maps known as brain maps. This facility allows topographical data to be readily compared and although this material is not shown here it was used extensively with the raw waveforms as an aid to analysis.

Results

Recordings were obtained from 13 normal control volunteers (six men and seven women; mean age 29.9 years, range 19–57 years) and 19 schizophrenic patients (12 men and seven women; mean age 35.5 years, range 17–68 years). There was a positive family history of schizophrenia in three patients. The average time since onset of first illness was 10 years (range 2–40 years) and patients had been admitted to hospital for an average of 59 months (range 3 months–24 years). All patients were currently receiving drug treatment.

In the control group, of the 26 possible recordings obtainable (right and left index fingers), 24 were actually achieved. Two recordings were lost from two individuals with right-hand dominance from whom no recognisable potentials to stimulation of the left index finger were discerned, but who subsequently showed normal responses to median-nerve stimulation.

In the schizophrenic group, of the possible 38 recordings, 29 were actually achieved. In four patients, no recognisable components to digital stimulation could be discerned in either hand. Each of these patients subsequently produced clearly recognisable potentials to median-nerve stimulation. One patient had received major injuries to his left hand in a machine accident which had resulted in the loss of his left index finger.

Initial subjective analysis of the waveforms obtained from the parietal area resulted in the recognition of the 'W response' described by Desmedt & Brunko (1980) and Desmedt & Cheron (1980) – the familiar P17-N20-P27-N35-P45 components obtained by median-nerve stimulation. Although mean peak latencies are longer for digital than median-nerve stimulation, we adopted the nomenclature for responses to the latter for ease of communication and comparison.

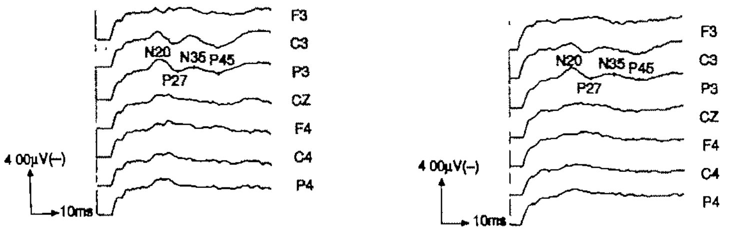
Table I shows the actual mean peak latencies of components of contralateral parietal hemisphere.

Other consistent components seen in both patient and control groups were frontal N17-P20-N30 and centroparietal N19-P22-N35-P45 potentials.

Utilising the software of the brain-mapping system, group average waveforms were calculated for the control group

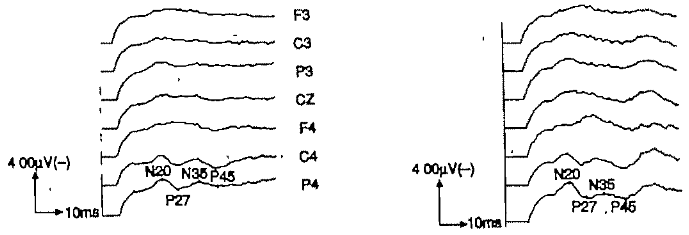
TABLE I
Mean peak latencies of components of contralateral parietal hemisphere in controls and schizophrenics with right and left index-finger stimulation

	Peak latencies: ms									
	P17		*N20*		*P27*		*N35*		*P45*	
	mean	*(s.d.)*	*mean*	*(s.d.)*	*mean*	*(s.d.)*	*mean*	*(s.d.)*	*mean*	*(s.d.)*
Controls										
Right index finger	17.0	(2.0)	22.8	(1.6)	29.4	(2.5)	37.7	(3.4)	46.8	(4.0)
Left index finger	17.1	(1.2)	22.3	(1.4)	28.2	(2.5)	36.1	(3.0)	44.6	(3.9)
Schizophrenics										
Right index finger	17.9	(1.7)	23.7	(1.7)	29.8	(1.8)	36.9	(5.6)	45.7	(4.5)
Left index finger	17.6	(1.0)	22.9	(1.4)	30.2	(2.6)	36.9	(3.6)	44.6	(3.8)



(a) (b)

FIG. 1 Stimulation of the right index finger: group mean data for controls (a) ($n=13$) and patients (b) ($n=15$).



(a) (b)

FIG. 2 Stimulation of the left index finger: group mean data for controls (a) ($n=11$) and patients (b) ($n=14$).

and schizophrenic patient group. The most relevant waveforms are illustrated in Figs 1 and 2. In order to identify any significant group differences, Student's *t*-test was performed using the brain mapper on every data point of every channel of the two groups. The subsequent results supported the subjective impression from the waveforms that there was no significant difference between the groups ($P>0.05$).

Peak latency (Table I) and peak-to-peak amplitude data (Fig. 3) were recorded from the waveforms obtained at electrode sites P3 and P4 for closer comparison with previous studies (Jones & Miller, 1981; Cooper *et al*, 1985). Statistical analysis using the *t*-test revealed no significant differences in any peak latency between patient and control group ($P>0.05$). Additionally, paired *t*-testing of interhemisphere peak latency revealed no significant differences of any parietal component ($P>0.05$). In both patient and control groups, the post-rolandic contralateral components were always of greater amplitude than their ipsilateral counterparts, with contralateral N20-P27 consistently the highest amplitude component for both groups. Comparison of the two groups was made by *t*-test of the group means of peak amplitude; for both right and left index-finger stimulation there was no significant difference in peak amplitudes between groups ($P>0.05$).

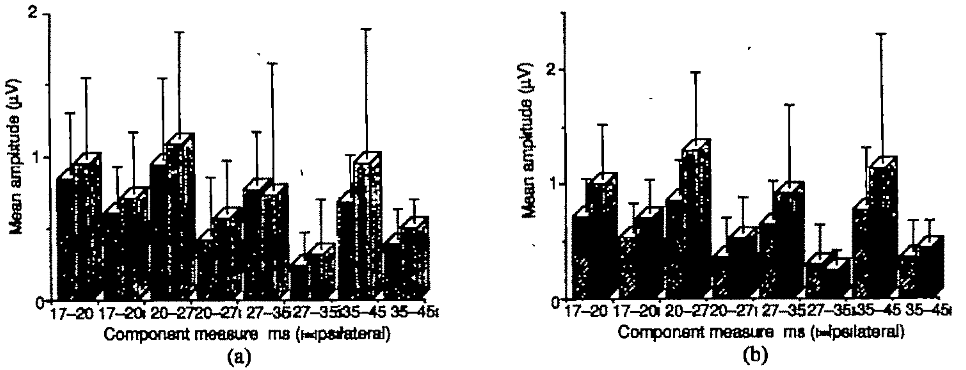


FIG. 3 Mean amplitude of parietal components in controls (■) and patients (▨); (a) right index-finger stimulation, and (b) left index-finger stimulation (error bars = 1 s.d.).

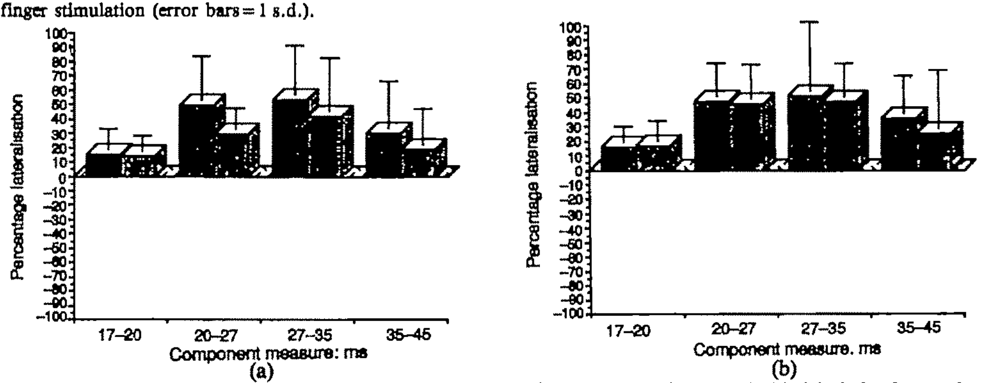


FIG. 4 Mean percentage lateralisation of parietal components in controls (■) and patients (▨); (a) right index-finger stimulation and (b) left index-finger stimulation.

In order to provide some objective assessment of the degree of lateralisation for each component, the following measure was used:

$$\frac{(\text{Contralateral amplitude} - \text{Ipsilateral amplitude})}{(\text{Contralateral amplitude} + \text{Ipsilateral amplitude})} \times 100\%$$

This formula provided a score which if positive (>0) indicated a degree of contralateral amplitude lateralisation and if negative (<0) indicated ipsilateral amplitude lateralisation. The mean percentage lateralisation for each component is shown in Fig. 4. There was no significant difference in lateralisation between the patient and control groups ($P > 0.05$). The mean lateralisation percentages were positive, indicating contralateral lateralisation for each component in both patient and controls.

At other scalp locations, centroparietal components (N19-P22-N35-P45) showed similar lateralisation features to those seen parietally in both patient and control groups. At frontal locations, components (N17-P20-N30) were bilaterally distributed in both groups. P17, although not clearly apparent on the group mean waveforms, was a consistently clear component in both patient and control groups. This component was recognisable in most recordings at every scalp location. These findings are in agreement with previous authors (Goff *et al*, 1977; Kakigi & Shibasaki, 1984; Desmedt & Cheron, 1980; Desmedt & Brunko, 1980; Duff, 1980).

One problem of comparing group mean data is that if an individual patient differed significantly from the control group, this fact may be lost when incorporated into a non-significant larger mean. To monitor for this, data from each schizophrenic subject were compared with the group mean of the control population using a Z-score technique utilising the Brain Atlas software in the manner of Duffy *et al* (1981), where:

$$Z = \frac{(x-m)}{s}$$

(x = individual subject data, m = control group mean data and s = control group standard deviation. Z is given in standard deviations from the control group.)

Each schizophrenic subject produced Z scores of less than two standard deviations across the entire waveform and at every scalp location with the exception of one subject. Patient SXI produced a waveform which on subjective inspection appeared to be quite bilaterally symmetrical across the entire waveform particularly at parietal locations (Fig. 5a). The subsequent Z score indicated a statistically significant deviation over the ipsilateral centroparietal and temporal areas. The highest area of deviation however appeared to be the temporal derivations. Re-inspection of the individual waveforms revealed a higher level of electromyelographic (EMG) artefact in the ipsilateral than the contralateral temporal region (Fig. 5b).

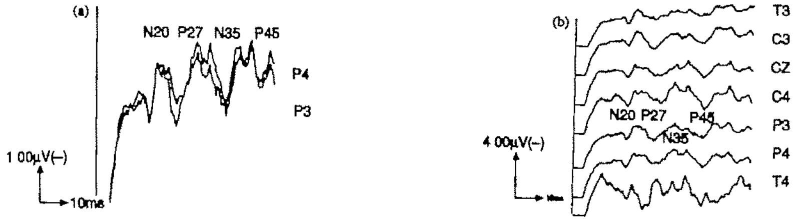


FIG. 5 Stimulation of the right index-finger of patient SXI: the waveform from parietal regions (a) is bilaterally symmetrical, while the waveform from the temporal region is asymmetrical as the result of EMG artefact.

Interpretation of the parietal component amplitudes without the benefit of knowledge of the extent of this asymmetric artefact could produce spurious conclusions. The effect of myogenic enhancement of the components on the ipsilateral hemisphere cannot be excluded (Bickford *et al*, 1964; Calmes & Cracco, 1971).

Discussion

Cooper *et al* (1985) felt that the nature of the stimulus employed in their study of schizophrenia was of major importance. Such a stimulus would achieve consistency with previous authors in this field who had employed either vibrotactile or similar mechanical stimulation (Jones & Miller, 1981; Tress *et al*, 1983). It was also felt that such a stimulus would be less distressing and more easily tolerated by schizophrenics than conventional electrical stimulation. However, there are a number of inherent problems in the design of vibrotactile speaker systems (Jones & Miller, 1981; Shagass *et al*, 1983; Cooper *et al*, 1985). A considerable audiostimulus is delivered to the patient which needs to be masked. The speaker cone is a low inertia system and subjective sensation is modified by pressure exerted from the stimulated digit. The duration of the stimulus is relatively long compared with constant current stimulation of electrical square-waves (10 ms v. 100 μs) and this may result in poorer formed potentials.

Most studies in the literature other than those investigating schizophrenics employ electrical stimulation to nerve trunk or digit, and it is therefore difficult to generalise from findings using the vibrating stimulus and to compare them with wider studies usually of larger groups of patients and controls. Cooper *et al* (1985) felt that their findings were probably stimulus-specific. However, since whatever mediated the ipsilateral cortical response of short latency (other than simple volume conduction) has to be the result of 'hardwired' thalamic and/or cortico-cortical tracts, the nature of the stimulation should be relatively inconsequential, unless we challenge existing anatomical, physiological and electrophysiological knowledge.

The correlation of SSEP abnormalities with proprioceptive deficits suggests that the SSEPs are mediated via dorsal column/medial lemniscal tracts (Halliday & Wakefield, 1963), with the small signals detected in lateral spinothalamic tracts appearing to contribute nothing to the scalp potential (Suzuki & Mayanagi, 1984). These facts coupled with existing knowledge of the cytoarchitecture of the primary somatosensory cortex lead us to conclude that cerebral locations of the SSEP are unlikely to differ widely, whether the stimulus is electrical or vibrotactile, if only digital nerves are stimulated (Hyvärinen, 1982; Kakigi & Shibasaki, 1984); the comparative morphology of tactile and electrical evoked responses would appear also to differ little (Pratt & Starr, 1981).

Given these factors and the relatively poor responses obtained on the vibrotactile stimulus, it was decided to standardise on electrical digital stimulation. It should be noted that the dominant components of Cooper's waveforms were positivities at approximately 30 ms and 45 ms which would equate well with the two dominant components obtained in this study, namely P27 and P45.

The final outstanding consideration was the tolerance of the electrical stimulus by the schizophrenic patients. During a pilot study, patients were able to tolerate both median and digital electrical stimulation at adequate levels (three times subjective current for digital stimulation and motor twitch of the thenar muscle for median nerve) for sufficient periods of time to allow adequate tracings to be obtained. Additionally, there were no topographical changes observed between stimulation rates of 0.7 Hz, 2.7 Hz and 4.7 Hz. These findings are broadly in agreement with those of Pratt *et al* (1980) who demonstrated that for both mechanically and electrically evoked SSEPs, stimulus rates of up to 4 Hz may be used without compromising the short-latency cortical components.

The results of the study are summarised as follows:

(a) There was no significant difference in either amplitude or morphology between the traces obtained from the control and patient group recorded from 20 scalp locations.

(b) There was no significant difference in peak latency comparing patient with control, and ipsilateral and contralateral latencies for right and left index-finger stimulation.

(c) The mean peak-to-peak amplitudes for parietal components were always greater for contralateral components compared with the ipsilateral counterpart.
(d) The percentage lateralisation quotient showed no lateralisation differences between the subject and control groups.
(e) When the data for each schizophrenic patient were compared with the control group data, no differences were found except in one case of asymmetric temporal EMG artefact.

The present study obtained adequate recordings of the SSEPs using a conventional digital nerve stimulus, and failed to replicate Cooper *et al*'s (1985) finding of a loss of lateralisation in a subgroup of schizophrenics. They tentatively hypothesised that their findings arose from abnormality in the corpus callosum. It has been established, however, that there are no direct callosal connections between the primary somatosensory hand areas in monkeys (Jones & Powell, 1969). Supporting evidence for this in man is provided by magnetic recordings to transcutaneous stimulation of the median nerve (Okada *et al*, 1984), which have revealed no ipsilateral components. Studies of regional cerebral blood flow (Foit *et al*, 1980) also have failed to reveal any ipsilateral components. This suggests that ipsilateral electrical components are volume conducted from the contralateral hemisphere. This is further supported by our lack of significant latency difference between ipsilateral and contralateral components.

An abnormality of the corpus callosum would not then allow one to predict theoretically a loss of lateralisation in the SSEP and our findings are therefore not surprising. It is disappointing that the present study was not able to confirm the presence of a neurophysiological marker of schizophrenia, as described by Cooper *et al* (1985). It does, however, raise a note of caution to experimenters examining lateralising events or topographical data generally, that careful monitoring of EMG must be undertaken. Unfortunately, recordings from the temporal region were not carried out during the study by Cooper *et al* (1985), so the question as to whether myogenic activity in the temporal region produced an apparently abnormal result for their schizophrenic group, has to remain unanswered.

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Extrapyramidal Signs, Primitive Reflexes and Frontal Lobe Function in Senile Dementia of the Alzheimer Type

D. M. GIRLING and G. E. BERRIOS

Of 146 elderly subjects suffering from Alzheimer-type dementia, 44% were found to have significant extrapyramidal signs. Although extrapyramidal signs were more common in those who had taken neuroleptic drugs in the preceding six months, 22 subjects (15%) who were drug free also had extrapyramidal signs. Scores for cognitive function and for 'frontal lobe' signs (verbal fluency, evidence of perseveration, and primitive reflexes) were found to correlate well with scores for extrapyramidal signs, suggesting that they reflect changes in a common substratum. It is tentatively suggested that this might be an abnormality in the dopamine system.

Patients suffering from Alzheimer's disease have a remarkable heterogeneity of clinical features. A number of attempts have been made to address this issue and to identify subgroups. For example, Bondareff (1983, 1987) has suggested the existence of two such groups: AD1, with insidious onset, gradual progression, little involvement of the locus coeruleus, limited symptoms, moderate ventricular enlargement, and affecting an older age group; and AD2, with rapid evolution, severe motor symptoms, marked ventricular enlargement, atrophy of the locus coeruleus, and affecting younger subjects. Berrios (1985) described a 'presbyophrenic' subtype characterised by elation, hyperactivity, marked memory impairment and preserved social graces, and suggested that it might be related to severe atrophy of the locus coeruleus.

Mayeux *et al* (1985), in turn, have reported four subgroups: (a) extrapyramidal (sporadic and drug induced), with severe intellectual and functional decline and frequent psychotic symptoms; (b) benign, with little or no progression; (c) myoclonic, with severe intellectual decline and frequent mutism, often with early onset; and (d) typical, with gradual intellectual and functional decline and no distinguishing features. The subgroup with extrapyramidal signs (EPS) has been described before. Pearce (1974) reported that about two-thirds of a sample of 65 patients with pre-senile dementia exhibited akinesia, rigidity, mask-like facial expression, or positive glabellar tap. Molsa (1984) confirmed this by finding that only 8% of a sample of 143 (neuroleptic-free) patients with Alzheimer-type dementia were free of EPS; the most common type of involvement being a rigid hypokinetic and hypomimic pattern. Dyskinetic signs were seen in 17%. Mayeux *et al* (1985) reported EPS unrelated to neuroleptic drugs in 34 (28%) Alzheimer patients, and no EPS in 13 (11%) who received no neuroleptic drugs. Patients with EPS performed worse on the modified Mini-Mental State Examination (MMSE). Interestingly, those with drug-induced EPS were even more impaired on the MMSE and on measurements of functional capacity.

The presence of EPS may reflect involvement of several neurotransmitter systems, or possibly a deficit in the dopamine system. The latter could account for the simultaneous presence of both EPS and some of the 'cognitive' deficits, although the role of the basal ganglia and dopaminergic tracts in cognition remains controversial. Some consider these structures as sustaining only motor behaviour, their only cognitive role being related to construction and execution of motor plans (Marsden, 1982). However, Cummings & Benson (1984) have suggested that they play a role in deficits related to arousal, attention, mood, motivation, memory, abstraction and visuospatial skills, and it has also been suggested (Taylor, 1986) that the dementia observed in Parkinson's disease results from disturbance of frontal lobe function caused by declining dopaminergic transmission. A deficit in the dopamine system could lead to frontal-lobe-type dysfunction, to release of some primitive reflexes, to soft neurological signs, and to symptoms described as constituting the so-called 'subcortical' type of dementia (Albert *et al*, 1974; Cummings, 1986) such as slowness of mental processing, forgetfulness, impaired cognition, and apathy. Snout and glabellar reflexes, visual perseveration and grasp reflex have been considered to be markers of ageing, and also an index of pathological change in the frontal lobe basal ganglia (Jenkyn *et al*, 1985; Bulbena, 1987). Some of these changes have also been found to be associated with severity of dementia in Alzheimer's disease (Tweedy, 1982; Huff, 1986).

Quinn (1986) has iterated, following suggestion by Rossor (1981), that Alzheimer-type dementia and Parkinson's disease may be related. In a clinicopathological study of 20 patients with Alzheimer-type dementia Ditter & Mirra (1987) found that extrapyramidal signs, especially rigidity, were related to coexisting pathology of Parkinson's disease. Patients with Parkinson's disease have been noted to have deficits involving memory, abstraction, visuospatial integration and central processing time (Pirozzolo *et al*, 1982; Mortimer, 1982; Freedman, 1986; Rogers *et al*, 1987; Politynska *et al*, 1990*a*). Furthermore, a correlation has been demonstrated between the degree of dementia and the severity of bradykinesia and rigidity (Martilla & Rinne, 1976; Mortimer, 1982; Mayeux *et al*, 1981). The view that the common substratum to these cognitive and motor symptoms may be dopamine deficiency, is reinforced by the finding that some of the intellectual deficits of Parkinson's disease improve with treatment with *l*-dopa (Marsh *et al*, 1971; Delis *et al*, 1982; Taylor *et al*, 1987).

In spite of the fact that the dopamine-deficiency hypothesis offers an economical explanation for the various ways in which EPS, cognitive deficit and frontal lobe dysfunction seem to combine in elderly people, little work has been done evaluating the three groups of signs simultaneously. If all three sets of signs are related to dopamine function, it can be hypothesised that they will correlate, and this effect would be present in all cases with EPS, regardless of whether or not they are related to neuroleptic treatment.

This project was designed to evaluate the association between the presence of EPS, measures of cognitive deficit, and markers of frontal lobe function (including primitive reflexes) in a sample of elderly subjects without clinical evidence of cerebrovascular pathology, and either with or without neuroleptic treatment.

Method

The sample comprised 146 elderly subjects in the city of Cambridge with a clinical diagnosis of Alzheimer's disease. To achieve as balanced a sample as possible, four different populations were sampled: (a) all referrals to the Psychogeriatric Department at Fulbourn Hospital over five months; (b) all admissions to the psychogeriatric-assessment and intermittent-relief wards at Fulbourn Hospitals over five months; (c) in-patients under the care of the Department of Geriatric Medicine at Addenbrooke's Hospital; (d) residents of all local-authority homes for the elderly within a ten-mile radius of Fulbourn Hospital. From interview with informants and review of medical records the history of the condition and past medical and psychiatric history were established. Subjects were included if they had: progressive deficit of intellect and memory for more than six months, resulting in impairment of social functioning. Subjects were excluded if they had: clinical evidence or history of stroke or Hachinski score of 5 or greater (Hachinski *et al*, 1974); a diagnosis of Parkinson's disease before the onset of intellectual decline; or evidence of metabolic, toxic, or traumatic cause of dementia. For a number of potential subjects the information available was felt to be inadequate to make a firm diagnosis and these subjects were excluded. Many of the residents of local-authority homes had been assessed by the Department of Geriatric Medicine or the Psychogeriatric Department and the quality of their records was good.

All assessments were carried out by DMG. In addition to the current and past medical and psychiatric history, background and demographic data, family and drug histories were obtained at interview and from review of records. Details of current behaviour were recorded and informants were asked specifically about: the subject's general level of motor activity (e.g. whether she/he wanders); aggression; any evidence of hallucinations or delusions; and evidence that the subject would repeat an activity many times after she had been asked to do something else. (The latter was taken to suggest perseverative behaviour and examples included a lady who would make her bed over and over again and was persuaded to stop and eat lunch with extreme difficulty, and a lady who would wipe the tables in the ward continuously in spite of all attempts to engage her in other activities.) The Dementia Rating Scale (Blessed *et al*, 1968) for change in performance of every-day activities and change in habits was completed from interview with an informant.

The protocol included: the MMSE (Folstein *et al*, 1975; Teng *et al*, 1987); a measure of verbal fluency (Miller, 1984); the Schonell graded word reading test (Nelson & O'Connell, 1978); the Webster scale (Webster, 1968); and standardised recordings of grasp reflex, visual perseveration, glabellar reflex, and snout reflex (see Appendix). Evidence of perseveration in answering questions was noted and combined with the behavioural observations described above to form the variable 'clinical perseveration' in the data analysis.

A physical examination was also performed and the presence of EPS was rated on the Webster scale. This is a clinical rating scale in which each of the following signs are rated for severity on a four-point scale (0 = no involvement, to 3 = severe involvement): bradykinesia of hands; rigidity; posture; gait; upper-extremity swing; tremor; facies; speech; and self-care. This gives a total score of 0–27. A cut-off on the Webster scale of five was chosen to define Parkinsonian 'caseness'. Those scoring higher than this figure obviously had EPS, and thus false positives were minimised, as cognitive impairment can contaminate some of the items of this scale.

Demographic and other clinical information was recorded on a standardised data sheet. The time taken to assess each subject (excluding informant interviews and review of the notes) was approximately 45 minutes. The pilot study showed this to be the maximum length of time for which it was possible to maintain subject co-operation and interest.

Ethical approval for the study was obtained from the district ethical committee. Data were analysed using the SPSS-X PC V2 statistical package on an IBM PC-AT.

TABLE I
Demographic and clinical features (n = 146)

	Home for elderly (n = 43)	*Psychiatric ward (n = 59)*	*Medical ward (n = 20)*	*Psychogeriatric out-patients (n = 24)*
No. of women/men	36/7	40/19	14/6	12/12****
Mean age (s.d.): years	85.1 (6)	80.0 (6.7)	86.8 (6.6)	79.4 (5.2)*
Mean onset age (s.d.): years	81.0 (6)	76.3 (7.4)	84.6 (6.5)	76.1 (5.7)*
Mean duration of dementia (s.d.): years	4.3 (2.5)	3.8 (1.9)	2.3 (0.9)	3.5 (2.3)*
Mean dementia score (doubled) (s.d.)	17.1 (6.8)	18.9 (6.0)	14.0 (3.7)	13.3 (4.6)*
Mean MMSE score (s.d.)	12.3 (6.3)	10.4 (5.9)	13.1 (3.8)	16.3 (4.8)*
Mean verbal fluency score (s.d.)	7.2 (6.1)	5.2 (5.8)	8.2 (6.2)	12.1 (4.8)*
Mean Schonell (pre-morbid IQ) (s.d.)	104 (11)	100 (11)	92 (7.4)	106 (10)
no. of patients tested	22	31	14	22
No. with probable family history of dementia (%)	0	14 (24)	0	4 (17)
No. co-operative with assessment (%)	33 (77)	38 (64)	17 (85)	23 (96)
No. retarded (%)	4 (9)	11 (19)	4 (20)	2 (8)
No. aggressive (%)	5 (12)	20 (34)	2 (10)	7 (29)***
No. with tendency to wander (%)	12 (28)	27 (46)	1 (5)	8 (33)*
No. with preserved social graces (%)	31 (72)	27 (46)	16 (80)	19 (79)*
No. with clinical perseveration (%)	3 (7)	14 (24)	1 (5)	3 (13)
No. on neuroleptic medication (%)	16 (37)	42 (71)	6 (30)	9 (38)
No. with positive grasp reflex (%)	4 (9)	7 (12)	1 (5)	8 (33)
No. with visual perseveration (%)	4 (9)	17 (29)	3 (15)	3 (13)
No. with positive glabellar reflex (%)	10 (23)	12 (20)	1 (5)	3 (13)
No. with positive snout reflex (%)	7 (12)	12 (20)	2 (10)	3 (13)
Mean Webster score (s.d.)	5.1 (4.6)	6.6 (5.4)	3.7 (3.6)	3.1 (4.6)**
range	0–14	0–19	0–12	0–14

*$P<0.00$, **$P<0.01$, ***$P<0.02$, ****$P<0.03$ (χ^2, Kruskall–Wallis).

Results

There were 102 women with a mean age of 82.4 years (s.d. 7), mean Blessed score of 16.5 (s.d. 6.5), mean MMSE score of 12.3 (s.d. 6.1), mean Webster score of 4.5 (s.d. 4.4), and mean verbal fluency score of 6.7 (s.d. 7.6); there were 44 men with a mean age of 82.1 years (s.d. 6.6), mean Blessed score of 17.4 (s.d. 5.3), mean MMSE score of 12.4 (s.d. 5.6), mean Webster score of 7.8 (s.d. 5.2), and mean verbal fluency score of 6.8 (s.d. 6.9). Seventy-three subjects had taken neuroleptics during the previous six months. There was a significant difference (Mann–Whitney $P<0.000$) between the sexes for Webster scores, which remained when corrected for receipt of neuroleptics. Table I shows values for all variables partitioned according to the four population samples. Significant differences were found in age and, predictably, in cognitive status and frontal lobe measures, with subjects in the psychiatric wards being the most, and those at home (the psychogeriatric out-patients) the least, affected.

Table II shows the effect of taking neuroleptics on frontal, cognitive and EPS variables. Predictably, those taking medication had significantly higher overall scores in EPS measures, but it is notable that 22 subjects had Webster scores over 5 in the drug-free group. An exploratory factor analysis was carried out (not shown in Table II) of Webster items of subjects according to neuroleptic status. For neuroleptic-free subjects the analysis yielded two factors: F1, including bradykinesia, facies, speech and self-care, and F2, including posture, gait, swing and tremor. This factor structure disappeared when the same analysis was carried out for the group on neuroleptics: F2 disappeared but for tremor (loading = 0.94).

From Table II it can be seen that all of the Webster items were endorsed by drug-free patients. Subjects in the psychiatric wards showed the highest incidence of EPS (most of them were also receiving neuroleptics). 'Frontal lobe' measures are also partitioned according to whether or not subjects had taken neuroleptics in Table II. Significant differences were found only for positive glabellar tap ($P<0.02$) and clinical perseveration ($P<0.05$).

Table III shows the significant correlations between cognitive, frontal and EPS measures. It was found that scores on the Webster scale were correlated with verbal fluency and MMSE scores, and with the functional dementia rating ($P<0.001$, two-tailed), suggesting that presence of EPS is associated with severity of dementia. EPS scores also correlated significantly ($P<0.01$, two-tailed) with presence of primitive reflexes (grasp reflex, glabellar reflex, snout reflex and visual perseveration) and with presence of perseverative behaviour.

Subjects were then separated according to whether they were Parkinsonian 'cases' and were taking neuroleptics, and compared on their endorsement pattern in the Webster scale by means of the Mann–Whitney test. No significant differences were found on any item except 'self-care'.

Exploratory principal-component analysis was carried out

TABLE II
Summary of measures according to drug status

	Taking neuroleptics *(n = 73)*	*Drug-free* *(n = 73)*
Mean dementia score (s.d.)	17.7 (5.7)	15.8 (6.5)***
Mean MMSE score (s.d.)	11.3 (5.9)	13.2 (5.8)
Mean Schonell (pre-morbid IQ) estimate (s.d.)	101 (11)	102 (11)
no. of patients tested	$n = 38$	$n = 51$
Extrapyramidal measures		
Mean Webster score (s.d.)	6.5 (5.2)	3.95 (4.3)*
No. with Webster score >5 (%)	42 (58)	22 (30)*
Mean bradykinesia score (s.d.)	0.71 (0.7)	0.47 (0.7)
Mean rigidity score (s.d.)	1.0 (0.9)	0.59 (0.8)*
Mean posture score (s.d.)	0.81 (0.8)	0.66 (0.77)
Mean gait score (s.d.)	0.89 (0.7)	0.73 (0.7)
Mean arm swing score (s.d.)	0.84 (1.0)	0.37 (0.7)
Mean tremor score (s.d.)	0.29 (0.5)	0.15 (0.4)
Mean facies score (s.d.)	0.67 (0.8)	0.33 (0.6)
Mean speech score (s.d.)	0.62 (0.8)	0.22 (0.5)
Mean self-care score (s.d.)	0.60 (0.6)	0.44 (0.7)
'Frontal lobe' measures		
Mean verbal fluency score (s.d.)	7.0 (7)	7.7 (6)
Mean score for soft neurological signs (s.d.)	0.7 (0.8)	0.5 (0.7)
No. with clinical perseveration (%)	16 (22)	5 (7)***
No. with positive grasp reflex (%)	6 (8)	7 (10)
No. with positive visual perseveration (%)	15 (21)	12 (16)
No. with positive glabellar reflex (%)	18 (25)	8 (11)
No. with positive snout reflex (%)	14 (19)	10 (14)

*$P<0.001$, **$P<0.01$, ***$P<0.02$, ****$P<0$ 05, (Mann-Whitney or χ^2 two-tailed tests).

using ten variables (age, sex, MMSE score, dementia score, verbal fluency, neuroleptics, soft neurological signs, Webster score, social graces, and sample population). Three factors were extracted with an eigenvalue >1, and accounting for 66% of the variance). They were named: 'dopamine factor' (which included loadings from cognitive, frontal lobe and motor measures), 'sex factor' (which included loadings from sex, EPS and population variables), and 'age factor' (which included loadings from age, neuroleptics, and population variables).

A discriminating function including coefficients for age, sex, frontal and cognitive measures, and neuroleptics classified 80% of the subjects correctly according to presence or absence of EPS. If the coefficient for neuroleptics was not included, 77% of the subjects remained correctly classified.

TABLE III
Variables significantly correlated with score on the Webster scale

Preserved social graces	0.65**
Combined measure of cognitive function	0.73**
Combined measure of 'frontal lobe' function	0.56**
Dementia score	0.59**
Verbal fluency	0.53**
MMSE score	0.49**
Aggressiveness	0.36**
Co-operative at interview	0.35**
Clinical perseveration	0.28**
Gender	0.35**
Neuroleptics	0.26*
Positive grasp reflex	0.28**
Positive glabellar reflex	0.39**
Visual perseveration	0.27*
Duration of cognitive impairment	0.22*

*$P<0.01$, *$P<0.001$, (two-tailed significance).

Discussion

This study tried to deal with the most common disadvantages of naturalistic studies. Patients were taken from various populations in order to include subjects with a range of disabilities. Measures of cognitive and functional impairment were used which are known to have clinical relevance. This was more difficult in the case of 'frontal lobe' dysfunction (Stuss & Benson, 1984; Wang, 1987), classic tests of which (e.g. Wisconsin Card Sorting Test and more recent modifications) are practically very difficult to use in a dementing population. Verbal fluency is a measure sensitive to frontal lesions that is relatively easy to administer and so used in an attempt to distinguish these two types of cognitive impairment.

Soft neurological signs and primitive reflexes have also been associated with 'frontal lobe' function, but their meaning remains equivocal. Primitive reflexes have been shown to have a low inter-rater reliability but the four with the better reliability (Bulbena, 1987) were chosen for inclusion in this study.

An inevitable problem in clinical studies of Alzheimer-type dementia relates to the initial diagnosis. Even when very strict criteria are used the diagnosis of Alzheimer's disease is notoriously unreliable being, to a certain extent, a descriptive diagnosis by exclusion (Sulka *et al*, 1983; Homer *et al*, 1988).

The sample collected showed considerable variation in degree of cognitive impairment, Parkinsonian symptoms and frontal lobe dysfunction. It was also balanced in the proportion of subjects who had taken neuroleptics in the preceding six months. Subject scores, when analysed by population sample, showed a consistent pattern of impairment, with those in a psychiatric ward exhibiting the worst, and those at home the best performance. It is of some interest, however, that grasp, snout, and glabellar reflexes and visual perseveration were equally distributed in the four populations. This might suggest that these signs are unrelated to the dementing process or that, if they are, they do not contribute to psychosocial incompetence. The use of the Schonell test, as a measure of pre-morbid intellectual function, proved unhelpful, as a number of subjects were unable to complete this measure, because of either visual or cognitive impairment.

The taking of neuroleptics correlated with clinical perseveration and, as expected, with Webster scores, but had no effect on the rest of the variables, particularly the measures of frontal lobe function. This would suggest that impairment of the latter does not depend upon the same dopamine receptor populations as motor Parkinsonian signs. The fact that the taking of neuroleptics correlated with clinical perseveration, however, might simply indicate that the latter was a marker of the behaviour which gave rise to the need for neuroleptics in the first place (i.e. severity of dementia; in fact perseveration and dementia score correlated, $r=0.23$; $P<0.03$).

Using our criteria for Parkinsonian 'caseness' (i.e. a score of greater than 5 on the Webster scale), 64 (44%) of the sample were Parkinsonian 'cases', of whom 42 (29%) had taken neuroleptic medication and 22 (15%) had not. Comparison of the endorsement pattern on the Webster items for these two last groups showed no difference except on 'self-care', which is a measure likely to be contaminated by cognitive impairment and other non-Parkinsonian pathology. Thus, on achieving clinical Parkinsonian 'caseness', subjects cannot be differentiated on the basis of whether or not they are taking neuroleptics, contrary to what Hausner (1983) has suggested.

Viewing the whole sample, however, significant differences were found according to the neuroleptic status of the subjects. Thus neuroleptic-free subjects, irrespective of whether they were Parkinsonian 'cases', had better scores on all Webster items except posture, gait and tremor. As indicated above, the exploratory factor analysis was able to differentiate a different pattern of EPS in subjects according to the neuroleptic status, with the neuroleptic-free subjects showing a pattern similar to that found in patients with Parkinson's disease (Politynska *et al*, 1990*b*).

Variables reflecting Parkinsonian, cognitive and frontal lobe impairment were found to correlate well. This effect was even more marked if composite variables were constructed for each group by adding the scores of individual variables together. The correlations were all >0.5 ($P<0.01$) (after Bonferroni correction). In spite of the fact that it is probable that these correlations have been magnified by item commonality, they are also likely to reflect changes in a common substratum. The strong association between the three types of impairment may reflect illness severity and the derangement of a number of different neurotransmitter systems; however, dysfunction of the dopaminergic system alone could provide the explanation. Bakchine *et al* (1989) reported independently a correlation between primitive reflexes, extrapyramidal signs and cognitive impairment in a sample of 91 patients suffering from dementia of the Alzheimer type.

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Appendix: Primitive reflex protocol

Grasp reflex

Each palm is stroked with moderate pressure from the wrists to the fingers. A positive response is any grasping motor movement of the hand. Absence of response of the subject's hand is normal.

Visual perseveration (in spoken-commands test)

This test attempts to reveal visual perseveration of the examiner's movements in spite of the verbal request to perform a mirror image movement. (a) Examiner takes his own left hand and touches his own right ear while asking the patient to "Take your left hand and touch your right ear". (b) Examiner takes his own right hand and touches his own left knee while asking the patient to "Take your right hand and touch your left knee". A complete mirror image on one side is considered positive. No mirror image is considered negative.

Glabellar blink reflex

Elicited by tapping between the eyebrows with the finger and looking for eyelid movement, avoiding a visual threat response, and asking the patient not to blink. Responses persisting after nine taps are scored as abnormal.

Snout reflex

Elicited by pressing firmly with the index finger over the philtrum of the upper lip, a pursing/pouting movement is considered a positive response.

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The Clinical Efficacy of Citalopram in Treatment of Emotional Disturbances in Dementia Disorders

A Nordic Multicentre Study

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In this multicentre study, the clinical efficacy of citalopram was investigated in 98 patients with moderate AD/SDAT or VD using a combined double-blind and open technique with placebo and citalopram. Analyses were made for each diagnosis after four weeks of double-blind treatment. Patients with AD/SDAT treated with citalopram showed a significant improvement in emotional bluntness, confusion, irritability, anxiety, fear/panic, depressed mood and restlessness. Those improvements were not found after treatment with placebo. There were no significant improvements in patients with VD. No improvements were recorded in motor or cognitive impairment. Citalopram provoked few and comparatively mild side-effects. None of the changes observed during the double-blind withdrawal period were identified as withdrawal symptoms or rebound phenomena.

It is well known there are serious disturbances of the serotonergic (5-HT) metabolism in the brains of patients with senile dementia of Alzheimer type (SDAT)/Alzheimer's disease (AD) and in the brains of patients with vascular dementia (VD). Post-mortem investigations have shown low concentrations of 5-HT and its metabolite 5-hydroxy-indoleacetic acid (5-HIAA) as well as reduced 5-HT-sensitive [3H]imipramine binding in certain brain regions of subjects with SDAT/AD (Gottfries *et al*, 1983; Hardy *et al*, 1985; Carlsson & Gottfries, 1986; Marcusson *et al*, 1987; Wallin *et al*, 1989).

Citalopram has been shown to block the uptake of 5-HT into serotonergic neurons, with little effect on the uptake of noradrenaline or dopamine (Hyttel, 1982). The pharmacokinetics of citalopram is characterised by high bioavailability after oral administration and a plasma half-life of 30–40 hours (Fredricson-Overö, 1978, 1982).

There are some preliminary reports on the clinical effect of drugs inhibiting the neuronal 5-HT reuptake in patients with dementia. Alaproclate (Bergman *et al*, 1983) was given to 12 patients with AD/SDAT. Ratings of the clinical efficacy showed a positive effect in the emotional function in five of the 12 patients. Harenko & Elgen (1984) treated 10 AD/SDAT patients with citalopram and found that the drug seemed to reduce some emotional symptoms and to increase the patients' activity level. In another study (Nyth *et al*, 1987), 23 patients with dementia were treated with citalopram. Significant improvements were found in motor impairment, impaired concentration, absent-mindedness, emotional blunting, fear/panic and restlessness. The results of these studies support the hypothesis that 5-HT reuptake inhibitors may especially improve emotional functions in patients with dementia disorders.

Until now, there has been no controlled study published of the efficacy of selective 5-HT reuptake blockers in the treatment of patients with dementia. This paper reports results of a double-blind multicentre investigation of the clinical efficacy of citalopram and placebo in patients with a moderate degree of either VD or SDAT/AD.

Method

The 98 patients with AD, SDAT and VD included in the study were stratified according to diagnosis and onset of illness. Table I shows the distribution of the patients according to diagnosis, age and sex. The patients were diagnosed according to DSM–III (American Psychiatric Association, 1980) criteria for primary degenerative dementia and multi-infarct dementia. Fifteen of the patients had different concurrent somatic diseases like artrial fibrillation, coronary disease, heart insufficiency, angina pectoris, hypertension, cholelithiasis, diverticulitis of colon, prostatic cancer, skin cancer of the face, varicose ulcer of the leg, cerebral apoplexia, diabetes mellitus, hyperglycaemia, pernicious anaemia, immunopathy pattern (in cerebrospinal fluid; CSF), Parkinsonism and gout. Some of the patients were under long-term treatment for their concurrent disease with appropriate drugs like diuretics, antihypertensives or vasodilators.

Patients with severe dementia were not included. To assess the level of dementia, the patients were rated with the Gottfries-Bråne-Steen (GBS) geriatric rating scale (Gottfries *et al*, 1982). This includes a number of items on which scores range from zero to six, where six implies maximal impairment. To be included, the patients had at least to be able to help in undressing and dressing themselves and in taking food (less than five points on items M1 and M2 of the GBS scale). The inclusion criteria further permitted only one of the following items to be scored six: orientation in space, orientation in time or personal orientation (items I1, I2 and I3 of the GBS scale). The maximum score of recent and distant memory (I4 and I5 of the GBS scale) had to be four, while the minimum score of recent memory had to be two (mild to moderate disturbance).

Patients who suffered from serious somatic illnesses (like cardiac decompensation and malignant arrhythmias, renal insufficiency, liver dysfunction, general gastrointestinal malabsorption, or disease of the haematopoietic system), those with a history of schizophrenia, epilepsy, alcoholism or drug dependence and those who had recently been treated with monoamine oxidase inhibitors were excluded.

Study design

The trial was carried out in Sweden at St Jorgen's Hospital in Gothenburg and at the Department of Geriatrics, General Hospital of Malmo, in Norway at the Department of Geriatric Medicine of Ullevål Hospital in Oslo and at Fyllingsdalen Nursing Home of Bergen, and in Finland at Sanerva, Kuopio, and Hatanpaä Hospitals. A combined double-blind and open technique with placebo and citalopram was used. Placebo was chosen as reference since there is no substance that is known to have any significant and specific impact on dementia disorders. Tricyclic antidepressants were not considered for use as reference because of their serious side-effects in elderly patients. The trial consisted of three periods of treatment (A, B and C) and was carried out over 16 weeks (Fig. 1). All patients were initially given placebo for one week (wash-out period). The patients were then randomly assigned to double-blind treatment with either citalopram or placebo during four weeks. This A-period was followed by eight weeks of treatment with citalopram as a known substance (B-period). The B-period was included for ethical reasons since we wanted to give all the patients the opportunity to be treated with active substance. Finally, the patients once more were randomly assigned to double-blind treatment during four weeks with either citalopram or placebo (C-period). The second randomisation was independent of the first one. The C-period was included to check withdrawal symptoms with a double-blind technique.

With regard to drug administration, the patients received tablets of identical appearance each containing either 10 mg citalopram or inactive substance. During the first two weeks of the treatment, two tablets (either 20 mg citalopram or placebo) were given daily at about 4 p.m. to each patient. If there was no satisfactory treatment effect at the beginning of the third week, the dose was increased to three tablets daily (either 30 mg citalopram or placebo). The dose was decreased to one tablet (either 10 mg citalopram or placebo) if there were troublesome adverse side-effects. There were no changes in medication if the therapeutic effect was satisfactory. At the beginning of the B-period the initial dose was 20 mg citalopram. After some days this dose could be changed according to the previous treatment or consideration by the doctor in charge to 30 or 10 mg citalopram. The same dose was then kept constant during the rest of the trial unless there were any adverse effects. Additional medication was allowed to treat concurrent somatic diseases. Necessary treatment with hypnotics and sedatives was also permitted.

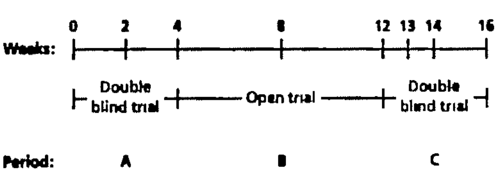

FIG. 1

Clinical investigations

The clinical efficacy of the treatment was assessed with different rating scales. Severity of illness was estimated on the Clinical Global Impressions scale (CGI; Guy, 1976), on which the scores range from one ('normal') to seven ("among the most severely ill"). Treatment effects were assessed on a global rating scale with scores ranging from one ('pronounced effect') to five ('deterioration'). The global assessments of the nursing situation were made on a six-point rating scale with scores ranging from zero ("needing almost no care") to five ("in need of complete care"). Symptoms, impairments and improvements were assessed with the GBS geriatric rating scale and the Montgomery-Åsberg Depression Rating Scale (MADRS; Montgomery & Åsberg, 1979). The GBS rating scale contains three different subscales measuring· motor impairment (six items, M1–M6); intellectual impairment (11 items, I1–I11); and emotional impairment (three items, E1–E3). There are also items describing six symptoms common in dementia (S1–S6). Each item is rated in seven steps ranging from zero (normal) to six (maximally impaired), where zero, two, four and six are pre-defined. The MADRS is a psychiatric rating scale measuring depression. It contains ten items, each of which is rated on a seven-point scale where zero, two, four and six are pre-defined.

The side-effects were assessed and recorded by means of the UKU side-effect rating scale (Lingjaerde *et al*, 1987). This scale is made up by 48 different symptoms. Both the symptoms themselves and the extent of the symptoms (range 0–3) are defined. For each side-effect the causal relationship to test treatment is evaluated.

The ratings were made before the treatment (– 1 week), at the end of the one-week wash-out period (baseline), and after two, four, eight, 12, 13, 14 and 16 weeks of treatment. In addition to these assessments, blood pressure and pulse were measured, and electrocardiogram (ECG) and laboratory tests were made once a month.

Biochemical investigations

In 15 patients (10 AD/SDAT and 5 VD), samples of CSF were taken before and after 8–12 weeks of treatment with citalopram. CSF (12 ml) was withdrawn by lumbar puncture under local anaesthesia at 8 p.m. The samples were immediately frozen and kept at a temperature of −70 °C until analyses. The CSF levels of HVA, 5-HIAA and HMPG were measured by gas chromatography/mass spectrometry according to a method described by Andersen *et al* (1981). The Ethical Committees of the various research centres and the national health authorities approved these biochemical investigations.

Statistics

Paired *t*-test was used to compare scores during treatment with baseline scores. The Mantel-Haenzel test (SAS, 1985) was used to compare distributions of improvements from baseline between groups for the single items of the GBS, the item severity of illness of the CGI and the assessment of the nursing situation.

Results

Of the 98 patients originally included in the trial, 61 completed treatment during 112 (s.d. 9) days. Nine patients (5 citalopram, 4 placebo) were withdrawn within two weeks (early withdrawals) and only baseline assessments were available. This left 89 patients for the efficacy analyses (Table I).

Table II shows, in addition to the number of early withdrawals, the number of patients who were withdrawn from the study later during the trial period (late withdrawals). The table also gives the reason for discontinuation of treatment. Three patients were withdrawn on day 1. One patient was withdrawn because of deterioration, the second because of tremor and the third because of leucopenia diagnosed on the basis of a blood sample drawn at baseline. The latter patient was the only patient who was excluded from the analysis of side-effects.

One patient was withdrawn on day 57 because of hemiplegia, one because of symptoms of influenza (day 31) and one patient died on day 26. The doctors in charge of the patients stated that these events were not related to citalopram treatment. One patient had ECG changes due to digitalis intoxication and the ECG normalised after reduction of the digitalis dose. Twelve patients were withdrawn from the study because the Norwegian authorities required more investigations in animals before this trial could continue. Two patients were withdrawn on days 101 and 109, respectively, because of increased confusion. These patients were assessed according to the protocol and were considered to have completed the study.

TABLE I
Distribution of patients according to diagnosis, age and treatment

	AD/SDAT	*VD*	*Total*
Sex			
male	11	9	20
female	56	13	69
Average age	77.2	78.5	77.6
Period			
citalopram	31	13	44
placebo	34	11	45
Total no. of patients	65	24	89

TABLE II
Distribution of and reason for early and late withdrawal during the trial

	Early: n		*Late:* n	
	Citalopram	*Placebo*	*Citalopram*	*Placebo*
Unrest	1			
Diarrhoea	1			
Exanthema		1		1
Tremor[1]	1			
Unknown		1	3	
Leucopenia[1]	1			
Increased serum alkaline phospatase		1	1	
Refusal		1	1	
Deterioration[1]	1		1	1
Regulatory claims in Norway			5	7
Dizziness			1	
Somnolence			1	
Confusion			2[3]	1
Hemiplegia[2]			1	
Died			1	
No effect			1	
Symptoms of influenza[2]			1	
ECG-findings[2]			1	
Total	5	4	20	10

1. Withdrawal on day 1.
2. The doctor in charge of the patient stated that the event was not related to citalopram treatment
3. Withdrawn on days 101 and 109.

The initial daily dose of citalopram in the A-period was 20 mg. After two weeks the dose was increased to 30 mg in 20 patients, 17 patients remained on 20 mg and one patient had his dose reduced to 10 mg a day. In the placebo group 14 patients received two tablets a day during the entire A-period and 28 patients had their dose increased to three tablets a day. Information about possible dose changes was missing for a few patients in both groups. In periods B and C most patients received either 20 mg or 30 mg a day.

Clinical investigations

The GBS ratings at baseline indicated emotional disturbances in 85 patients. Forty-six patients had one to three disturbances of emotional functions while 39 patients had four or more disturbances recorded. The most common disturbances were reduced motivation ($n = 62$), emotional

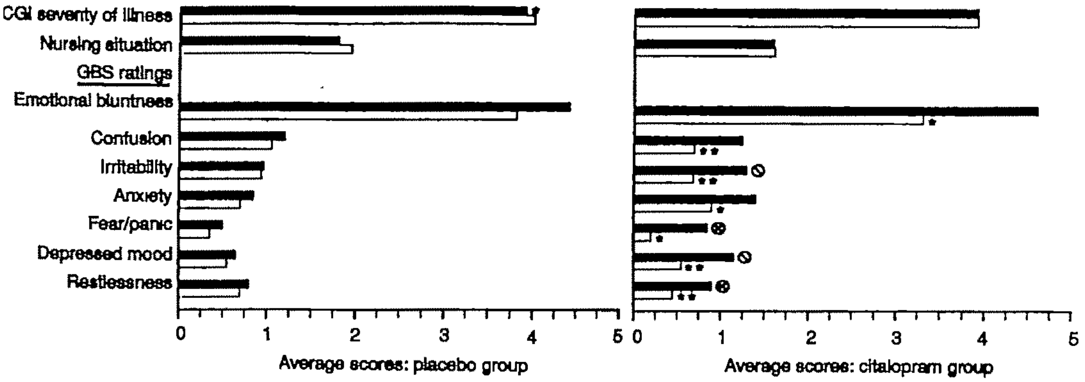

FIG. 2 Average scores in patients with diagnosis AD/SDAT at baseline (■) and at week 4 (□) after treatment with double-blind technique (*$P<0.05$, paired t-test; **$P<0.01$, paired t-test (within groups); ⊘ $P<0.05$, unpaired t-test (between groups); and ⊗ $P<0.10$, unpaired t-test (between groups).

TABLE III

Average rating scores in patients with diagnosis AD/SDAT at baseline and at week 4 after treatment with double-blind technique

	Citalopram group *Average score*			*Placebo group* *Average score*			*Improvement: between group differences*		
	Baseline	*Week 4*	n	*Baseline*	*Week 4*	n	T	*d.f.*	P
CGI-severity of illness	3.897	3.897	29	3.909	4.032*	33	1.087	35[1]	0.284
Assessment of nursing situation	1.607	1.643	28	1.795	1.971	34	0.809	45[1]	0.423
GBS-ratings									
Subscale 1: motor impairment	7.408	6.667	27	8.282	7.813	32	0.346	48[1]	0.731
Subscale 2: intellectual impairment	22.666	21.333	27	20.063	19.875	32	1.001	51[1]	0.321
Subscale 3: emotional bluntness	4.555	3.296*	27	4.406	3.781	32	0.877	57	0.384
Item 21 confusion	1.223	0.704**	27	1.188	1.063	32	1.465	57	0.148
Item 22 irritability	1.297	0.667**	27	0.969	0.938	32	2.463	57	0.017*
Item 23 anxiety	1.408	0.889*	27	0.876	0.688	32	1.100	57	0.276
Item 24 fear-panic	0.852	0.185*	27	0.500	0.344	32	1.900	30[1]	0.067
Item 25 depressed mood	1.149	0.519**	27	0.657	0.563	32	2.361	29[1]	0.025*
Item 26 restlessness	0.888	0.444**	27	0.782	0.719	32	1.777	57	0.081
MADRS total score	8.307	6.115*	26	7.690	6.897	29	0.935	28[1]	0.358

*$P<0.05$ (paired t-test for within group differences; two-tailed t-test for between group differences)

**$P<0.01$ (paired t-test, within group differences). d f. computed according to the algorithm of Smith-Satterthwaite.

1. Group variations differ significantly at the 20% level (two-tailed test)

blunting ($n=45$), anxiety ($n=38$), and confusion ($n=37$). The MADRS ratings showed only mild depression in most patients with a mean total MADRS score of 8.1 (s.d. 6.6). Two patients had a mean total MADRS score above 26, indicating the presence of major depression.

For the A-period, the data analysis was performed for each subgroup separately. Because of the small number of patients in each subgroup (see Table I), we chose to present the results of the data analysis of the subgroups in two larger groups consisting of (a) patients with AD/SDAT and of (b) patients with VD. Because of missing data, the number of patients is not quite the same for all rating scales.

The global assessments of severity of the illness on the CGI showed a small but significant deterioration at week four for the placebo-treated AD/SDAT group (Table III and Fig. 2). There were no improvements recorded in the VD group.

When the status at baseline was compared with the status after four weeks of double-blind treatment, the AD/SDAT patients treated with citalopram showed significant improvements in emotional bluntness, confusion, irritability, anxiety, fear-panic, depressed mood and restlessness. No significant changes were recorded in any of the GBS items in the placebo-treated patients during this period (Table III and Fig. 2).

Table III and Fig. 2 also show comparisons in GBS ratings between the placebo-treated AD/SDAT group and the citalopram-treated AD/SDAT group. Irritability and depressed mood were significantly more improved at week four in the citalopram-treated patients and the difference in fear-panic and restlessness bordered on significance for citalopram. Table III also shows that neither motor performance nor intellectual functions were influenced by the treatment.

No significant changes were recorded in the VD group during treatment with either citalopram or placebo in the A-period.

In the AD/SDAT group treated with citalopram, mean total scores of MADRS ratings were significantly reduced at the end of the A-period ($P=0.02$). No significant changes were recorded in the AD/SDAT group treated with placebo. In the VD group no significant changes were shown during this period. There was no deterioration in any of the MADRS variables during the period.

With regard to the B-period, the initiation of citalopram treatment in the former placebo-treated AD/SDAT group was not associated with the same improvements as were found after treatment with citalopram at week four. At week eight there was a significant improvement in emotional bluntness. However, this improvement was not found at week 12. The observed improvements in the citalopram treated AD/SDAT patients at week four remained during the B-period. In the VD group there were no significant changes during the B-period.

The analyses during the C-period were done disregarding diagnoses. The period was added with the sole purpose of studying possible withdrawal effects under double-blind conditions. A new randomisation of the patients was thus done at the beginning of the C-period. However, no symptoms of withdrawal effects or rebound phenomena were seen. The ratings on the different rating scales indicated that there were some deteriorations as well as some improvements in various variables during this final phase of the study.

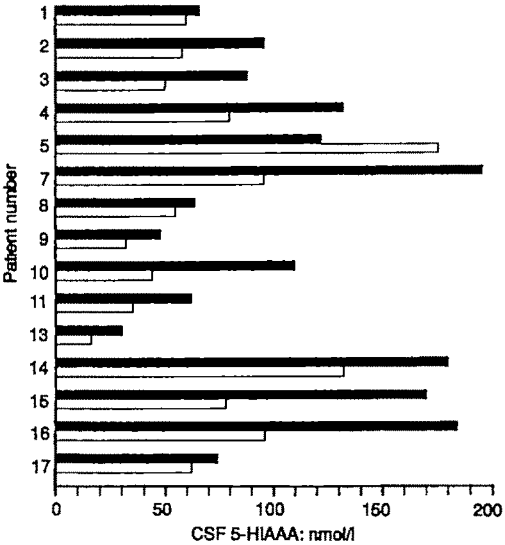

FIG. 3 Concentrations of 5-HIAA in cerebrospinal fluid in 15 patients at baseline (■) and after 8–12 weeks (□) of treatment.

Biochemical investigations

Fig. 3 shows the results of the investigations of CSF. 5-HIAA showed, as expected, a decrease at the end of the B-period ($P<0.05$). The ratio between 5-HIAA and HVA was significantly decreased ($P<0.01$).

Side-effects

Ninety-seven patients were included in the analyses of side-effects. In citalopram-treated patients no or only a few side-effects were recorded. The global assessment of side-effects showed no significant difference in these between citalopram- and placebo-treated patients. Eleven symptoms recorded on the UKU side-effect rating scale were rated as probably induced by test treatment. These were recorded in 10 patients treated with either placebo ($n=3$) or citalopram ($n=6$). The tenth patient had the same side-effect, increased fatigue, both during placebo and citalopram treatment. Four of the side-effects – sleepiness, increased duration of sleep, rash, and increased fatigue – were recorded both during treatment with citalopram and with placebo. Concentration difficulties, depression, emotional indifference, orthostatic dizziness, diminished sexual desire, ejaculatory dysfunction and orgastic dysfunction were recorded only during treatment with citalopram. Most symptoms were mild or moderate. Only ejaculatory dysfunction ($n=2$) and orgastic dysfunction were assessed as degree three. Except bradycardia in three patients with an initial heart rate at 48, 52 and 60 respectively, no clinically significant cardiovascular reactions or blood pressure changes were recorded.

Discussion

This double-blind study indicates that emotional disturbances in patients with AD/SDAT improve when treated with citalopram, but not with placebo. It is well known that depression in the elderly is difficult to diagnose. One may thus wonder whether depression may have been present in the patients to a greater extent than the MADRS scores indicate. The MADRS ratings showed only mild depression with an average total MADRS score of 8.1. Two patients had a total score above 26, indicating the presence of major depression. The GBS ratings at baseline showed depressed mood but also other emotional disturbances in 85 of the 97 patients. Other studies also suggest that emotional disturbances are common in patients with dementia disorders (Bråne & Gottfries, 1985; Reifler *et al*, 1986; Merriam *et al*, 1988; Andersson, 1989). We regard these emotional disturbances, depression included, as part of the dementia syndrome which have so far been overlooked. Emotional bluntness, confusion, irritability, anxiety, fear/panic, depressed mood, and restlessness were improved after four weeks of treatment with citalopram. These improvements were not evident in the patients treated with placebo. No significant improvements were found in the VD group which, however, was smaller ($n = 24$) than the AD/SDAT group ($n = 65$). No effect was recorded on motor and intellectual impairment.

An unexpected finding was that the symptom of confusion significantly improved during the citalopram treatment. This finding is difficult to explain and the clinical importance of it must be explored. One may speculate that the confusion was due to depressed mood; relieving the depression alleviated the confusion. However, our clinical impression was that the two syndromes were not related.

The initiation of citalopram treatment in the former placebo-treated group was not associated with the same improvements as were found after treatment with citalopram at the end of the double-blind period. Perhaps the investigators were expecting a more pronounced improvement after eight to twelve weeks in the trial and thus smaller improvements may have been overlooked.

None of the changes recorded during the withdrawal of citalopram could be identified as withdrawal symptoms. It is of importance to note that the status of the patients who completed the whole study (16 weeks) did not deteriorate significantly from the status at baseline. Karlsson *et al* (1988) have shown that a significant progression of the dementia illness can be expected during a period of only two months. In this study a similar progression could not be demonstrated after four months. This absence of progression of the illness may be due to drug treatment.

The biochemical investigations showed, as expected, a significant decrease in 5-HIAA in the CSF. This has been found in studies by Bjerkenstedt *et al* (1985) and Balldin *et al* (1988). The decrease of 5-HIAA is interpreted as a feedback inhibition of transmitter synthesis and release due to reuptake blockade of the transmitter (see Bjerkenstedt *et al*, 1985).

The therapeutic effect was evaluated in 89 patients. Sixty-one patients completed the whole treatment period, 24 discontinued treatment prematurely during treatment with citalopram (five early withdrawals and 19 late withdrawals) and 13 discontinued treatment prematurely during treatment with placebo (four early withdrawals and nine late withdrawals). In all, treatment with citalopram constituted 75% of the whole treatment period. In relation to this there is no difference in the relative number of patients who discontinued treatment during placebo and citalopram. The reason for withdrawal was in no case due to any serious complication that could be related to the citalopram treatment. Citalopram was thus found to provoke few side-effects in this study. In the global assessment of side-effects there were no significant differences between citalopram- and placebo-treated patients.

Our finding that citalopram induces few and rather mild side-effects is in agreement with studies of citalopram in depressed patients (Danish University Antidepressant Group, 1986; Shaw *et al*, 1986; Gravem *et al*, 1987). Also, this finding seems to be the same as that seen in younger depressed patients. The few side-effects seen were not dose-related. Several of the elderly demented patients had concurrent somatic disorders like cardiovascular diseases or diabetes. No cardiovascular reactions or blood pressure changes were recorded with the exception of bradycardia in three patients with a low heart rate at baseline. This is in accordance with findings in other studies (Lindegaard-Pedersen *et al*, 1982; Christensen *et al*, 1985). Based on these results, citalopram in doses between 10 mg and 30 mg must be considered a drug provoking few side-effects when administered to elderly patients, even if these patients have concurrent cardiovascular diseases, diabetes or other diseases and are under treatment with other drugs for this reason.

This is the first controlled study reporting that a selective 5-HT reuptake inhibitor seems to have a beneficial effect in patients with dementia disorders. Further studies are needed to answer the question of whether the magnitude of the improvement is of clinical significance. Studies in groups of patients selected for emotional disturbances are needed to elucidate the efficacy of the citalopram treatment. However, we would like to stress that it was originally the relatives of the patients who drew out attention to the possible benefits of citalopram. After the pilot study (Nyth *et al*, 1987) many relatives wanted us to continue the treatment because it facilitated daily dealings with the patient. We had the same reaction from relatives after this controlled study. Since emotional disturbances are common and are most troublesome for the demented patients themselves and for their relatives, this study suggests that citalopram may be valuable in the treatment of elderly demented patients with AD/SDAT.

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A Tribute to Lasègue's Description of Anorexia Nervosa (1873), with Completion of its English Translation

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Gull and Lasègue are usually credited with the first description of anorexia nervosa. In general, however, the work of the French neuropsychiatrist has been underestimated, and English-speaking colleagues who refer to Lasègue's writing on 'hysterical anorexia' almost exclusively rely on the translation of his French paper, which in 1873 appeared in a British medical journal, the same year as the original publication. However, some important passages had been omitted from the translation, and this has not previously been noted.

Long-lasting food refusal, unusual fasting and peculiar hunger strikes have been described for many centuries (Brumberg, 1988; van Deth & Vandereycken, 1988). But the history of morbid self-starvation as a clinical entity (anorexia nervosa) begins only around 1873. One of the central figures is Sir William Withey Gull (1816–90), the renowned and fashionable London physician, who from the very beginning strongly claimed that he had been the first to discover the new syndrome. As we have discussed in detail elsewhere (Vandereycken & van Deth, 1989), a close examination of the events clearly demonstrates that one should attribute discovery of anorexia nervosa to both Gull and the French neuropsychiatrist Lasègue. While Gull (1874) uses the typical nosological style of the physician, enumerating the principal symptoms and findings, Lasègue gives a colourful picture, with astute psychological observations. The Frenchman's paper should be regarded as a classic in psychiatric literature, not just because of its historical place, but also for its astonishing insight.

Life and work of Lasègue

Born in the same year as Gull (5 September 1816), Ernest Charles Lasègue did not earn the same reputation as his British colleague (Hanot, 1883; Ritti, 1885; Streletski, 1908; Semelaigne, 1932; Morel, 1983), but he ranks without a doubt as one of the great French psychiatrists of the mid-19th century, together with Bénedict-Augustin Morel (1809–73) and Valentin Magnan (1835–1916). He was overshadowed, however, by the famous Jean-Martin Charcot (1825–93), the 'Napoleon of the Neuroses', whose work on hysteria and hypnotism at the Parisian hospital La Salpêtrière inspired to a great extent the thinking of Sigmund Freud. This, together with some English chauvinism, might explain why his name is so often misspelled in Anglo-Saxon literature. The most frequent misspelling as Lasèque is probably due to Bliss & Branch (1960, p. 13 ff.), whose chapter on the history of anorexia nervosa had been for a long time the only major source for English-speaking colleagues. The fact that Lasègue was not as renowned as Gull or Charcot does not make his scientific contributions less important. His list of publications is quite impressive (see Ritti, 1885; and the collected works: Lasègue, 1884) and embraces internal medicine, psychiatry, neurology, and the history of medicine. Beside his article on hysterical anorexia, he was the first to describe exhibitionism and folie à deux, the latter together with Jules Falret (1824–1902). Other important psychiatric writings concern delusions of persecution, catalepsy, kleptomania, melancholy, different forms of hysteria, and various articles on alcoholism.

Lasègue was first trained as a teacher in philosophy and rhetoric. In 1838 he was appointed to the prestigious Lycée Louis-le-Grand in Paris, where the poet Charles Baudelaire (1821–67) was one of his pupils. The young teacher soon made friends with Claude Bernard (1813–78), the pioneering physiologist, who persuaded him to start studying medicine. Lasègue's academic career developed more slowly. Invited to the Salpêtrière by Claude Bernard, who worked there under the direction of Jean-Pierre Falret (1794–1870), Lasègue became passionately involved with the study of medicine in general and psychiatry in particular. Having acquired his doctoral degree in medicine in 1846, he soon became fascinated by his work as consultant physician at the Prefecture of Police, where he had to examine an endless number of people accused of all sorts of crime. Here he developed his special style of work: he kept standardised notes of all cases, and when he intended to describe some common pattern of abnormal behaviour, he collected all his case notes and wrote an article about it. Out of this forensic work arose many of his pioneering descriptions. Thus it is no wonder that he was recognised as an authority in medicolegal issues. As Gull in 1853 became full professor, Lasègue's academic career was just beginning (with the acceptance of his Aggregation Thesis on general progressive paralysis), but the following year he was appointed editor of the *Archives de Médicine Générale*, the influential journal in which he would publish most of his articles. In 1862 he was nominated Lecturer in Nervous and Mental Diseases, in 1867 he became Professor of General Pathology and, finally, in 1869 he took up the prestigious Chair of Clinical Medicine in La Pitié hospital, which he occupied until his death. Lasègue was not intent upon titles or honours, and although elected a member of the French Academy of Medicine in 1876, instead he remained devoted to his clinical work and to his many teaching functions at the Faculty of Medicine in Paris. He suffered from severe diabetes and died at the age of 67 on 20 March 1883.

Lasègue seemed to be a calm person of inconspicuous appearance who carefully weighed his words. He was much loved by his colleagues and highly esteemed by his students, who were impressed by his eloquent lectures. In scientific circles he was known as a shrewd sceptic. His previous training in philosophy and rhetoric probably influenced a great deal of his further career in neuropsychiatry. Although inspired by Bernard's work, he always critically questioned the fundamentals of medicine and was averse to any kind of dogmatism. His life and work did certainly share one important characteristic with Gull's: the observation of facts as the cornerstone of scientific endeavour. Sufficiently acquainted with the most important English and German literature, he knew the history of neuropsychiatry quite well. But he himself was not eager to have his name connected with some major scientific discovery. In fact his name was given to the 'Lasègue sign' – raising the extended leg while the patient is lying down, to provoke sciatic pain – but one of his students had, in his doctoral thesis of 1881, described and named the test (see Bazzi & Donadi, 1964; de Sèze, 1958).

How keen an observer he was, can be concluded from the paper we wish to discuss here.

The English translation (part I)

In April 1873 Lasègue published an article, "De l'anorexie hystérique", in the *Archives Générales de Médecine* (the article has been included in the 1884 collection of his medical writings, vol. I, pp. 45–63; a reprint may also be found in Corraze (1971), pp. 135–150). Soon after its publication, the paper must have caught the attention of one or more British physicians. Barely five months later, on 6 and 27 September of the same year, a two-part English translation entitled "On hysterical anorexia" was published in the *Medical Times and Gazette* (this article is reprinted in Kaufman & Heiman (1964), pp. 143–155, and for the most part also in Andersen (1985), pp. 19–27, and Harrison & McDermott (1972), pp. 567–571). Translations of French articles were then not uncommon and remind us that the British academic world was not isolated from the Continent.

The anonymous translation is in general quite good but somewhat loose. Besides giving Lasègue an acute instead of a grave accent, the most important defect is the omission of several parts of the original text. As far as we know, nobody seems to have noticed this – even Skrabanek (1983) in his otherwise detailed and reliable historical study believes that Lasègue's French text was translated *in toto*. Only in one place in the translation is an omission noted by the translator, while four other passages have been omitted without indication (see Appendix).

Lasègue began his article by underscoring the importance of his contribution:

> "The object of this memoir is to make known one of the forms of hysteria of the gastric centre which is of sufficient frequency for its description not to be, as too readily happens, the artificial generalisation of a particular case, and constant enough in its symptoms to allow of physicians who have met with it controlling the accuracy of the description, and to prevent those who have yet to meet with it in their practice being taken unawares."

Then the author depicts the process of the illness as if he were referring to one case (he characterises it as "a somewhat diagrammatic sketch of the disease"). This is the typical style of Lasègue's psychiatric writings, as he tries to give a 'prototypical' description of the disorder. In the final paragraphs of the article, Lasègue emphasises once again the uniformity of the case histories:

> "The cases which have served me as a basis for this memoir are eight in number, all women, the youngest being 18, and the eldest 32. . . . Although these cases are few in number, they so much resemble each other that the latter ones found me in no indecision in regard either to diagnosis or prognosis, and, in fact, all passed on according to rule." (1873*b*, pp. 368–369)

Addressing "a symptomatic complexus too often observed to be a mere exceptional occurrence," Lasègue characterises his article as a contribution to the nosology of the numerous "disturbances of the digestive organs which supervene during the course of hysteria" (1873*b*, p. 265). In this context, the choice of the term 'hysterical anorexia' is not surprising:

> "The term 'anorexia' might have been replaced by 'hysterical inanition', which would better represent the most characteristic of the accidents; but I have preferred the former term, without otherwise defending it, precisely because it refers to a phenomenology which is less superficial, more delicate, and also more medical." (p. 265)

Lasègue then puts forward his major hypothesis about the origin of the lack of appetite he observed in these cases: they abstain from food "to avoid pain, which, although hypothetical, is dreaded in advance" (p. 265). It usually concerns a young woman, between 15 and 20 years of age, who "suffers from some emotion which she avows or conceals". So, in Lasègue's view, the origin (the 'primary cause') is a psychic one leading to some vague suffering ('gastralgia') after food intake. Then starts what may be called the 'learning process' in the development of the disorder: "The patient thinks to herself that the best remedy for this indefinite and painful uneasiness will be to diminish her food". And so she does and, after a few weeks of refusing food, "the disease is now declared" (p. 265).

Then follows a strong warning, which even today may be given to many general practitioners and internists: "Woe to the physician who, misunderstanding the peril, treats as a fancy without object or duration an obstinacy which he hopes to vanquish by medicines, friendly advice, or by the still more defective resource, intimidation" (p. 265). To avoid these 'medical faults', Lasègue knows how important the differential diagnosis of gastric pain is, and he devotes three paragraphs to a detailed description of its characteristics. But the key to the diagnosis should be searched for in the patient's attitude:

> "The hysterical subject, after some indecision of but short duration, does not hesitate to affirm that her only chance of relief lies in an abstinence from food; and, in fact, the remedies appropriate to other gastralgias are here absolutely inefficacious, however zealously both physician and patient may employ them." (p. 266)

At this point in the translated text, a first passage has been deleted (Appendix (a)), including the mention of a crucial diagnostic clue: the 'paradoxical liveliness' of the anorexic, a characteristic that is stressed repeatedly throughout the further text. In fact, Lasègue elaborates on this theme in the same paragraph. He states that the first weeks or even months may proceed "without the general health seeming to be unfavourably influenced".

> "There is no emaciation, although the amount of nutriment scarcely amounts to a tenth of that habitually required by the patient. The power of resistance of the general health in the hysterical is too well known for astonishment being excited at seeing them support without injury a systematic inanition to which robust women could not be exposed with impunity. Moreover, this diminution of aliment is made not suddenly, but by degrees, so that the economy more easily habituates itself to the decrease." (p. 266)

The next sentence has been deleted in the translation (Appendix (b)), although it contains a historically interesting remark which explains why Lasègue was so impressed by the sometimes astonishing liveliness and hyperactivity of the anorexics, regardless of their state of severe malnutrition. Thus the anorexics' behaviour stood in sharp contrast to the appearance of fellow citizens during the famine in Paris due to the German siege in the winter of 1870–71, during the Franco-Prussian war. As Schadewaldt (1965, p. 3) notes, Lasègue with his medically trained eye could clearly see the difference in behaviour between the self-starved anorexics and the forcibly starved Parisians. As the text continues (after the deleted part):

> "Another ascertained fact is, that so far from muscular power being diminished, this abstinence tends to increase the aptitude for movement. The patient feels more light and active, rides on horseback [the French text also mentions: 'she takes long walking-tours'], receives and pays visits, and is able to pursue a fatiguing life in the world without perceiving the lassitude she would at other times have complained of." (Lasègue, 1873*b*, p. 266)

In the next paragraph, Lasègue turns to another important element in the further course of the disorder: "the dispositions of those surrounding her". He first stresses the naïvety of doctors who believe in a rapid cure with medicines: the patient will take them all, but remains "invincible in regard to food".

> "When after several months the family, the doctors and the friends perceive the persistent inutility of all these attempts, anxiety and with it moral treatment commences; and it is now that is developed that mental perversion, which by itself is almost characteristic, and which justifies the name which I have proposed for want of a better – hysterical anorexia."

Then follows a striking depiction – a brilliant piece of observation – of a *family scene* one can still perceive:

> "The family has but two methods at its service which it always exhausts – entreaties and menaces – and which both serve as a touchstone. The delicacies of the table are multiplied in the hope of stimulating the appetite; but the more the solicitude increases, the more the appetite diminishes. The patient disdainfully tastes the new viands, and after having thus shown her willingness, holds herself absolved from any obligation to do more. She is besought, as a favour, and as a sovereign proof

> of affection, to consent to add even an additional mouthful to what she has taken; but this excess of insistence begets an excess of resistance." (p. 266)

Do not expect any concession from these patients, adds Lasègue, for this makes all the difference between 'obstinate hystericals' and 'capricious children'. With this lively and somewhat ironic passage ends the first part of the English translation.

The English translation (part 2)

The second part of the translation did not appear in the next issue of the *Medical Times and Gazette*, but three weeks later (in weekly medical periodicals like this, it was usual to publish longer articles as a series). The text continues with Lasègue's further elaboration of what he considers to be a "most positive law that hysteria is subject to the influence of the surrounding medium". He emphasises how, for both patient and entourage, ideas and sentiments become narrowed, to such an extent that "the responses become still more uniform than the questions" (1873*b*, p. 367). In the next sentence, starting a new paragraph, the translator missed a short remark (Appendix (c)): as though in anticipation of the reader's doubt of what has been said so far, Lasègue stresses that his picture of the anorexic's family scene is far from exaggerated. He then sums up the patient's typical excuses for not eating. Finally, she always gets her way and the parents acquiesce: "The patient willingly joins her family at meals, on the condition that she is allowed to take only what she wishes" (p. 367).

Now Lasègue points to the characteristic attitude of the patient – "a condition of contentment truly pathological" – which stands in sharp contrast to other forms of anorexia. One would call it now the almost pathognomonic denial of illness: "Not only does she not sigh for recovery, but she is not ill-pleased with her condition, notwithstanding all the unpleasantness it is attended with". The anorexic shows

> "an inexhaustible optimism, against which supplications and menaces are alike of no avail: "I do not suffer, and must then be well," is the monotonous formula which has replaced the preceding, "I cannot eat because I suffer". So often have I heard this phrase repeated by patients, that now it has come to represent for me a symptom – almost a sign." (1873*b*, pp. 367–368)

In Lasègue's opinion, "the whole disease is summed up in this intellectual perversion", but he hastens to add: "Moreover, I do not believe that gastric hysteria is any exceptional occurrence, for in other hysterical localisations we meet with at least an equal indifference, however inconvenient and painful their manifestations may be" (p. 368). With this remark, the author takes the opportunity to discuss some other forms of hysteria. Here, a long passage has been skipped (Appendix (d)), as indicated by the translator: most of it is indeed irrelevant to the subject, except for the last paragraph, which describes the patient's obstinate resistance against treatment.

The text continues with a description of the third stage of the disease: "Emaciation makes rapid progress, and with it the general debility increases" (p. 368). Lasègue enumerates the typical signs – amenorrhoea, constipation, dry skin, and anaemia. "The appearance of these signs, the import of which can escape no one, redoubles anxieties, and the relatives and friends begin to regard the case as desperate." This sudden switch from a somatic to an interactional description, must have been surprising to Lasègue contemporaries, for he feels the need to explain it. For a present-day psychiatrist, familiar with systems theories, the following remark may seem superfluous, but it surely was a revolutionary statement in the eyes of 19th-century doctors:

> "It must not cause surprise to find me thus always placing in parallel the morbid condition of the hysterical subject and the preoccupations of those who surround her. These two circumstances are intimately connected, and we should acquire an erroneous idea of the disease by confining ourselves to an examination of the patient. Whenever a moral element intervenes in a disease, as here it does without any doubt, the moral medium amidst which the patient lives exercises an influence which it would be equally regrettable to overlook or misunderstand." (p. 368)

Substitute 'psychic' for 'moral', and the quote could have been taken from a modern psychiatric publication!

In this third stage of the disease, the "unconscious change in the respective positions of the patient and her friends" opens the way towards treatment:

> "The young girl begins to be anxious from the sad appearance of those who surround her, and for the first time her self-satisfied indifference receives a shock. The moment has now arrived when the physician, if he has been careful in managing the case with a prevision of the future, resumes his authority."

But one should expect and accept an understandable ambivalence on the part of the patient, for whom now two courses are open: "She either is so yielding as to become obedient without restriction, which is rare; or she submits with a semi-docility, with the evident hope that she will avert the peril without renouncing her ideas and perhaps the interest that her malady has inspired" (p. 368). Hence, the doctor knows that both this attitude and the sometimes long duration of illness will turn any therapeutic attempt into a process of trial and error.

This brings Lasègue to a discussion of the patient's prognosis. On the one hand, he is not very optimistic and underscores that "we should be on our guard against affirming beforehand the amount of amelioration with which we must rest content". On the other, he affirms that he has "never yet seen an anorexia terminate directly in death. . . . Hysteria, whatever extreme violence it may attain, is not itself mortal, but it may become the occasional or indirect cause of fatal diseases; and first among these is pulmonary tubercle" (p. 368). In the French text then follows a rather confusing passage (last missing part in the translation – Appendix (e)). Lasègue briefly gives the example of a married woman, 30 years of age, whose anorexia first appeared to him as a disguised suicide attempt, a hypothesis he soon had to abandon: ". . . the patient belatedly attempted to cling to life". This end is surprising because on the one hand it suggests that she did not want to die, but on the other it contradicts Lasègue's previous remark that he had never encountered a death due directly to anorexia. Perhaps the translator had the same difficulty with this passage and, therefore, decided to leave it out completely without indication.

Also somewhat in contradiction to the cautious opinion about prognosis he expressed above, Lasègue states: "The hysterical anorexia itself is always cured more or less completely at the end of years, passing through the period of decrease with an appetite that is limited or exclusive, and occasionally fantastical" (p. 368; the French text mentions 'bizarre' appetites). He illustrates this with some examples, among whom figures a woman with an 'invincible anorexia', whom he had treated, together with Trousseau (his late professor of medicine with whom he worked as *chef de clinique* and under whose guidance Lasègue published several of his early papers on internal medicine). Finally, Lasègue notes he had "never known the disease relapse". For some reason he expected to hear from recovered patients the real motive that had driven them to such extreme abstinence from food, but "the typical formula employed during the course of the disease was reproduced – 'I could not; it was too strong for me, and more, I was very well'" (p. 368).

Comments

The original article represents a superb piece of observation. From a psychological viewpoint it is superior to Gull's (1874) account. The London physician, however, apparently was so impelled to claim priority in describing the 'new' disease that he did not rate Lasègue's contribution at its true value (Vandereycken & van Deth, 1989). To our best knowledge Lasègue himself never referred to his own article in later publications. In 1881, in a lecture on appetite, he briefly mentions (as a special example of lacking appetite):

> "the hysterical anorexia, characterized by indifference, disgust, aversion, finally by satiation. The subject doesn't nourish herself anymore, her health diminishes and sometimes, rarely it is true, may end in death. . . . She condemns herself to a complete inanition, similar to that observed in some insane persons who reject for some reason all foods served for them. The matter is so mental in this young girl, though she is far from insane, that if one wants to overcome her refusals, one should be able to treat her by addressing the mental state itself. One should change the direction of her ideas and nourish her by means of a dissimulated feeding." (Lasègue, 1881*a*, p. 11)

That same year, another lecture – on the lies and stories hysterics can fabricate – starts with some remarks about the many, very confusing and vague signs of hysteria. Lasègue then briefly mentions three examples of vomiting and/or food refusal. Although at least the first one suggests a form of hysterical anorexia, the term is not used (Lasègue, 1881*b*, p. 111).

The picture Lasègue's biographers give is of a rather humble man, unlike Gull. Nowhere do we find any indication that he regarded his contribution as something special. Two anecdotal references support our view. In an Austrian medical journal of 1880, there is a short report (Anonymous, 1880) about his visit to some Parisian hospitals. In the department of Professor Lasègue he was shown "two cases of involuntary protracted fasting". Although he describes the clinical picture of these hysterical women, no word about hysterical anorexia is said, not even with regard to differential diagnosis. This is rather striking since the reporter discusses some unusual forms of self-starvation and quotes some French authors. Thus, apparently, Lasègue did not tell his visitor about 'his' disease nor about his publication about the subject! And in 1883, Deniau mentions in his doctoral thesis that Lasègue had seen more than 200 cases of hysterical anorexia. Although a highly improbable number, it indicates that Lasègue continued to see many anorexics without publishing anything further about them. These findings could also be explained by the enormous fame of Charcot, as already mentioned.

Charcot appears to hold the clue to two puzzling questions the content of Lasègue's paper aroused: why did he not remark on the desire for slimness of his patients, and why did he not recommend a treatment? With regard to the first question, it is indeed striking that both Gull and Lasègue did not notice the drive for thinness or the obsession with slenderness now considered a central diagnostic element in these patients. A careful analysis of the 19th-century literature on anorexia nervosa shows that several authors (especially in France) did mention weight concern as an important causal factor. Charcot emphasised this fear of becoming overweight to such an extent that, according to an ironic comment from Pierre Janet (1859–1954), it became an *idée fixe* (van Deth & Vandereycken, 1988; Habermas, 1989). Lasègue, on the contrary, probably overlooked this psychological factor because of his own theoretical bias, focusing too much on the 'gastric' hysteria: at a time when all kinds of digestive complaints like dyspepsia and epigastric pain ('gastralgia') were popular notions, he was easily misled by anorexics who used this 'disease language' to rationalise the self-starvation and emaciation they accomplished for aesthetic purposes (Shorter, 1987).

The second question was touched upon in 1904, when the French physicians Albert Mathieu and Jean Charles Roux wrote:

> "It is curious that Lasègue did not have recourse to a treatment which suggested itself from his very description of the illness. He had established that the milieu in which the patient lived is the proper ground for the development of the anorexia and it did not occur to him to separate the patient from her family." (Mathieu & Roux, 1904, p. 123)

Again, it was Charcot's influence that propagated this therapeutic principle of 'parentectomy' (Vandereycken *et al*, 1989). What became known as 'isolation' was applied for many decades to all kinds of psychiatric disorders, especially hysteria and neurasthenia (Camus & Pagniez, 1904). Hence, for both the weight concerns and the isolation one might imagine that Lasègue did not feel the need to correct and complete his previous opinion because Charcot had stressed these factors. As a matter of fact, some of Charcot's pupils tried to please their master by connecting his name to the disease *anorexie nerveuse* (Féré & Levillain, 1883), but, fortunately, the succeeding generations of French physicians did justice to Lasègue, although they replaced his term by the notion of *anorexie mentale* still in use today.

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Appendix

A comparison of Lasègue's original text (1873*a*) with the English translation (1873*b*) shows that five passages were not translated.

(a) Thirteen lines (1873*a*, p. 391) are omitted from p. 266 (left column, last paragraph) of the translation: "But another kind of circumstance, different from those which usually predominate in hysteria, should be mentioned. The patient has lost the sensation of appetite and, to get her to eat, she must overcome the fear of pain without being solicited or even encouraged to do so by the appetising quality of the food: her abstinence, on the contrary, satisfies two inclinations at the same time. All hysterical manifestations, if they were present, are suspended from that first phase onward. Far from becoming feeble or sad, the patient manifests a kind of alacrity unusual to her. One almost might say that she is taking her precautions for the times to come and that she prepares arguments which she will not fail to make use of."

(b) From the same p. 266 (right column, ninth line) the following remark (1873*a*, p. 392) is missing: "We all had this experience during the siege of Paris, noting that the diet forced upon the poor did not in any way have a major impact upon their health."

(c) From p. 367 (right column, before the first sentence of the second paragraph) the following sentence (1873*a*, p. 394) is missing: "Nevertheless, all those who shared these painful family scenes will realise that this tableau is certainly neither overdrawn nor too gloomy."

(d) From p. 368 a long passage (two and a half pages) has been skipped (1873*a*, pp. 396–398): Lasègue first gives an example of a 20-year-old girl who complained of pain while singing and speaking, and ended in complete mutism. Another hysterical patient (a 16-year-old girl) claimed she could not walk nor stand up because of muscular pains. It is not a simple question of exaggerating pain sensations (as in the case of 'imaginary invalids') In the case of hysteria, pain is but one symptom among many others Lasègue then returns to the subject of his article: "During this second period established as such, the patient remains the same: lack of appetite, fear of an ill-defined sensation, and an absolute and growing refusal even to try to nourish herself. The obstinacy lasts for months, if not years. In one case, in which I succeeded one of our masters as physician, the patient had received conscientious care for eighteen months. With a nonchalance mixed with a slight acidulity she would rehearse twice daily the same unvarying conversation with her physician: My child, have you finally made up your mind to eat? – Doctor, I've done what I can but it hasn't worked. – Just try a little bit harder and everything will be alright."

(e) Ten lines (1873*a*, p. 401) are skipped from p. 368 (right column, second paragraph): "In one case alone, a married woman with a long history of hysteria whose anorexia began at 30, I did observe the transformation I mentioned before The disgust for food was the result of some griefs more imaginary than real, but nonetheless profoundly felt. At first I suspected a concealed attempt at suicide. The further course forced me to abandon that supposition, as the patient belatedly attempted to cling to life."

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Psychotic Depression Presenting as Status Epilepticus

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A case of status epilepticus was secondary to water intoxication, which in turn was secondary to a depressive psychosis.

Water intoxication occurs in approximately 50% of psychiatric patients who compulsively drink water, the majority (80%) of whom have schizophrenia (Jose *et al*, 1979). Thus most reports have focused on water intoxication in schizophrenic subjects in whom this potentially fatal syndrome occurs as a late complication. This has been described as the 'pre-terminal stage' by Arieti (1945), developing on average 12–13 years after first psychiatric contact and characterised by diminution in acute symptoms and the development of primitive habits (Vieweg *et al*, 1984). Water intoxication is also associated with other features of chronic or residual illness such as tardive dyskinesia, cerebral ventricular enlargement, poor response to neuroleptics. Previous reports have also suggested that in the majority of cases polydipsia occurs in response to delusional beliefs (e.g. to drown a parasitic worm, to propitiate the Gods, etc.; Singh *et al*, 1985). In addition, the actions of some psychotropic drugs, including tricyclic antidepressants, lithium and major tranquillisers, have been implicated in the aetiology (Illowsky & Kirsh, 1988).

Reports of water intoxication occurring in subjects other than chronic psychotic patients are rare, although a few cases of uncomplicated compulsive drinking have been described in neurotic subjects (e.g. Silber, 1984). In addition, Lee *et al* (1989) have recently reported the case of a 16-year-old Chinese girl who compulsively drank water in order to induce a state of altered consciousness. Apart from this addiction she had no other psychiatric illness and successful treatment consisted of simple advice to stop the activity.

We have only found three previous case reports (Singh *et al*, 1985) which describe water intoxication in patients with affective disorder and in all cases hyponatraemia developed as a late complication, occurring several years after the onset of illness and in subjects who had other disabilities such as personality disorder, poor social or occupational functioning. The case we present adds to this small literature on water intoxication in affective disorder and is also atypical and striking for a number of other reasons. Firstly, hyponatraemia-induced status epilepticus was the patient's first psychiatric presentation although she admitted to previous minor episodes of depression which had failed to come to medical attention. Secondly, our patient's stated reason for drinking excessively were not delusional but were in response to poor appetite. Thirdly, she had not been taking any psychotropic medication. Lastly, she made a complete recovery from the episode and, unlike previously described psychotically ill cases, showed no deterioration of functioning at follow up.

Case report

EK, a 58-year-old married woman with two children had never been admitted to hospital until December 1987 when she gave up her part-time job in the local post-office because of non-specific chest pain. She was admitted for investigation of possible cardiac pathology. However, although noted to be pale and tired, all investigations were normal and she was discharged from hospital two days later.

She was admitted for the second time in July 1988 in status epilepticus. Renal investigations on admission gave the following results: in serum, sodium 117 mmol/l, potassium 3.9 mmol/l, chloride 85 mmol/l, urea 2.2 mmol/l, creatinine 71 μmol/l, osmolality 241 mOsmol/kg; in urine, osmolality 429 mOsmol/kg, sodium 87 mmol/l, potassium 47 mmol/l, urea 114 mmol/l.

All other investigations including full blood count were normal. She was resalinated and regained consciousness after 14 hours. Twenty-four hours after admission, she was fully orientated and serum electrolytes also reverted to the normal range. Subsequent investigations including liver and thyroid function tests, gamma-glutamyl transpeptidase, urine culture and computerised tomography headscan, barium meal and chest X-ray were all within normal limits, as was the electroencephalogram. Renal response to water loading was not assessed.

When interviewed by the duty psychiatrist she complained of marked loss of appetite and had lost weight (4.5 kg in three months). To compensate for her poor appetite and to ensure adequate hydration, she had started drinking large quantities of water and tea. For three months before admission her fluid intake had gradually increased although precise details of this were not available either from the patient or her husband. Her husband did report, however, that she was drinking almost continually for two days before the first seizure. Thus her fluid intake was relatively constant and showed no fluctuation. She gave a six-month history of early morning waking, anhedonia, poor concentration and gloomy thoughts concerning the future. She was not actively suicidal but did admit to feeling that life was not worth living. She had numerous physical complaints including pain in her back, arms, chest and abdomen. She had become increasingly withdrawn and easily confused and was unable to do the shopping or housework without her husband's assistance. She admitted to previous brief episodes of low mood over the past 10 years. However, these had been quite mild and had never caused her to stop work or seek treatment but were associated with an increase in fluid intake, although never previously to the degree on this occasion.

Her birth and upbringing in west Wales were unremarkable and she had moved to the Rhondda valley at age 15 years on account of her father's job as a Methodist minister. She left school unqualified and married her husband, a physics teacher, at the age of 19 years. Both her younger brother and her son had received psychiatric help for alcohol dependence. Her three older sisters have no psychiatric problems and both parents were deceased at the time of her illness. There was no family history of epilepsy and the patient herself had not previously had any seizures.

Pre-morbidly, EK described herself as self-confident, outgoing and someone who led a full and enjoyable life. She neither drank alcohol nor smoked. Her financial circumstances were secure, although her husband had lost a large proportion of his retirement 'lump sum' in the stock market crash of October 1987. During her illness, this fact had greatly concerned her.

Fourteen days after admission, she was transferred for psychiatric treatment. On arrival, she had little recollection of events leading to her admission and at interview appeared tense and agitated. Her speech was spontaneous and appropriate but rather abrupt in character with short sentences and minimal information given in response to questions. Her mood was depressed and thought-content included preoccupation with her illness, financial problems and the stigmatising effect of being a psychiatric patient. She expressed sensitive ideas of reference and persecutory ideation in connection with the comments she had made to the relatives of another patient, while on the medical ward. Following this she felt that the staff were talking about her and that she and her husband were going to be sued by these relatives and by the health authority. These ideas subsequently became delusional in intensity but rapidly responded to medication. She was correctly orientated and her memory was intact, although her performance on serial 7s and reverse months of the year was poor. She had limited insight into the psychiatric nature of her illness.

A diagnosis of psychotic depression was made and treatment with amitryptiline (100 mg nocte) commenced. Trifluoperazine (5 mg b.d.) was added and amitryptiline increased following the development of persecutory delusions. She responded rapidly to this regime and within four weeks of admission, her mental state had returned to normal. She was followed up as an out-patient for six months after which she was discharged without medication and symptom free.

An interview using the Schedule for Clinical Assessment of Neuropsychiatry (SCAN; Wing, 1988) was carried out one week after discharge and 10 weeks after presentation. Symptoms were scored for the episode of illness. The results are as follows: CATEGO-4 class R+, Index of Definition 8 (Wing & Sturt, 1978); tentative ICD-9 classification 296.2 (World Health Organization, 1978); ICD-10 diagnosis F31.0, severe depressive episode with psychotic features and mood congruent delusions (World Health Organization, 1988); DSM-III-R 296.24, major depression, single episode with psychotic features (mood congruent delusions) (American Psychiatric Association, 1987).

Discussion

We believe this to be the first case reported in which depressive illness has presented with status epilepticus induced by compulsive water drinking.

There have been three previous reports of the occurrence of self-induced water intoxication in depression (Singh *et al*, 1985), but the diagnosis of depression has always ante-dated the development of water intoxication and has developed in subjects with personality disorder or chronic illness.

EK stated that she was drinking excessively in a response to poor appetite and a wish to remain adequately hydrated. She complained of feeling very dry and having a choking feeling which she sought to alleviate by drinking. However, despite close questioning there was no evidence of any delusional belief system underlying her decision to drink excessively, and her explanation seems entirely plausible.

Eighty per cent of patients who develop water intoxication have schizophrenia but this is clearly not the diagnosis here. Although EK developed some persecutory delusions, these occurred late in the course of this episode, were not a prominent part of her illness and responded rapidly to treatment. A structured interview carried out just before discharge showed that the diagnosis was one of psychotic depression according to three different classifications, ICD-10, DSM-III-R and CATEGO-4.

In conclusion EK is clearly unusual in that she is not the type of chronic disabled patient with end-stage illness that is most commonly associated with water intoxication. Indeed, she remains a well integrated member of her local community. Her presentation was dramatic and life threatening but she made a complete recovery, and she was discharged from out-patient follow-up, and was free from symptoms and medication six months following admission.

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Leucopenia Secondary to Carbamazepine Despite Concurrent Lithium Treatment

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A manic–depressive patient developed leucopenia as a result of carbamazepine therapy, despite concurrent administration of lithium.

Haematological complications of carbamazepine therapy are well recognised (Pisciotta, 1975). Leucopenia is the most frequently reported haematological side-effect (Hart & Easton, 1982). In contrast, lithium is known to cause a leucocytosis (Shopsin *et al*, 1971), and it has been used in the treatment of agranulocytosis (Boggs & Joyce, 1983). Hence its effect should theoretically oppose the myelosuppressant effect of carbamazepine. We report a case of a 37-year-old man who developed leucopenia because of carbamazepine therapy despite concurrent lithium administration.

Case report

A 37-year-old male was admitted in September 1988 with mania (ICD–9, 296.0; World Health Organization, 1978). He was on lithium carbonate (1000 mg nocte) and his serum level was 0.82 mmol/l. He had a three-year history of manic–depressive psychosis and had been commenced on lithium in September 1987 after two previous episodes of mania which had required admission to hospital. His serum lithium in October 1987 was 0.67 mmol/l.

He required, however, readmissions in February 1988 and in August 1988, with further episodes of mania. His serum lithium concentration in February was 0.94 mmol/l and in August was 0.72 mmol/l. His episode in August was followed by three weeks of depression.

Family psychiatric history revealed a sibling had also been diagnosed as having manic–depressive psychosis.

On admission in September 1988, he was overactive, restless and had been spending money extravagantly. He had grandiose ideas but was not deluded. Therapy was commenced with haloperidol (10 mg t.d.s.) orally and the lithium was continued. His mood settled slowly but by three weeks he was euthymic. In view of the recurrence of his hypomania, despite adequate lithium treatment, it was decided to commence him on concurrent carbamazepine as these two drugs are a potent combination in the prophylaxis of manic-depressive psychosis. This was started on 18 October 1988 (100 mg b.d.). A white-cell count measured one week before this was 10.1×10^9/l and two weeks before this was 6.8×10^9/l.

The white-cell count was measured two days after the commencement of carbamazepine and this also was 6.8×10^9/l. His serum lithium level at that time was 0.71 mmol/l. On 25 October, the carbamazepine was increased to 200 mg b.d. and a serum level done two days later was 5.6 mg/ml. His serum lithium was 0.47 mmol/l on the same day. On 2 November, the carbamazepine was further increased to 200 mg t.i.d.

Five days later on 7 November he developed a pyrexia of 38.5 °C with a frontal headache, rigors and general malaise. He had no meningism, respiratory symptoms nor urinary symptoms. A white-cell count was performed and this was 5.6×10^9/l. Four days later on 11 November, his temperature had persisted and his white-cell count was 2.6×10^9/l with a differential of 60% neutrophils and 27% lymphocytes. In view of the leucopenia the carbamazepine was stopped. Blood culture, mid-stream urine specimen and immune screen performed at this time were all normal. A bone-marrow aspirate and biopsy was performed which revealed a reduction in the mature myeloid series, consistent with a drug-induced effect. After stopping the carbamazepine, the white-cell count rose to 3.1×10^9/l the following day and on 15 November was back to normal at 6.6×10^9/l. He was feeling very well, was apyrexial and his mood was euthymic. It was thus concluded that his fever and rigors were due to a viraemia and that the leucopenia was secondary to carbazepine therapy.

Discussion

This case is of particular interest because the leucopenia secondary to carbamazepine occurred despite the continuous administration of lithium. Haematological complications are well recognised with carbamazepine. Leucopenia, anaemia, neutropenia, eosinphilia, thrombocytopenia and aplastic anaemia have all been reported (Pisciotta, 1975). However, depression of bone marrow occurs much more frequently than a proliferative effect on the marrow and the commonest complication is leucopenia (Hart & Easton, 1982). The leucopenia may be either transient or persistent. It is estimated that a transient leucopenia occurs in approximately 10% of patients, usually in the first month of treatment (Livingstone *et al*, 1974). Carbamazepine may be continued and the leucopenia resolves spontaneously. However, with persistent leucopenia, the white-cell count will only respond to stopping carbamazepine (Gerber *et al*, 1979). The carbamazepine should be discontinued if the white-cell count is less than 3×10^9/l or if infection occurs (Hart & Easton, 1982). So far no discernible relationship has been found between dose of carbamazepine and leucopenia.

Lithium, used extensively in manic–depressive psychosis, has also been used in the treatment of agranulocytosis (Boggs & Joyce, 1983). It is known to cause a leucocytosis (Shopsin *et al*, 1971). Hence, one might expect it to counteract the marrow suppressant effect of carbamazepine. This did not happen in our case report. Our patient developed a serious leucopenia with a white-cell count of $2{\cdot}6\times10^9$/l and a fever with carbamazepine. It necessitated the stopping of carbamazepine. The patient made a complete recovery but this episode confirms that serious leucopenia may still occur with carbamazepine despite simultaneous lithium administration.

This is important to note as both these drugs are likely to be combined with increasing frequency in order to stabilise the affect of patients with recurring manic–depressive psychosis.

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Audiotape Therapy for Persistent Auditory Hallucinations

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We report a case of a man with recurrent depression and persistent second-person auditory hallucinations telling him to kill himself. Using an audiotape cassette and headphones the duration of the hallucinations decreased significantly. Helpfulness of the audiotape continued at 15 months follow-up

Auditory hallucinations are a source of considerable discomfort and are present in a variety of psychiatric patients. They have been associated with increased suicidal risk, at least among schizophrenic patients (Falloon & Talbot, 1981). Neuroleptic medication is the most common treatment, but the hallucinations persist in some patients. Patients try to decrease the voices by: (a) a change in posture (lie down, walk, run), activity (work, leisure such as music or television), interpersonal contact (withdrawing or engaging), and medication (increasing it or taking illicit drugs or alcohol); (b) attending to the task at hand; and (c) altering physiological arousal by such techniques as relaxation, reducing sensory input by the use of earplugs and closing eyes, or increasing arousal with exercise (pacing) and/or loud music to drown out the voices (Falloon & Talbot, 1981).

Psychological treatments of auditory hallucinations in schizophrenic patients have included occluding one ear and sound/noise delivered via headphones. In case studies auditory hallucinations decreased markedly when an ear plug was inserted in one ear (Birchwood, 1986; James, 1983; Done *et al*, 1986; Morley, 1987). Frequency and clarity of auditory hallucinations reduced with the use of headphones to deliver varying types of sound including music (Feder, 1982; Morley, 1987), white noise (Fonagy & Slade, 1982) and music, speech and white noise (Margo, 1981). The efficacy of this method might be related to the meaning of the sound for the patient (Margo, 1981).

In the literature we could find no studies of psychological treatment of depressive hallucinations. We report an encouraging case study of audiotherapy in a man who had recurrent depressive psychosis and troublesome second person auditory hallucinations commanding him to kill himself.

Case report

A retired 60-year-old security guard had been a day- and in-patient for over two years because of repeated depression with a serious suicidal risk. He had a 40-year history of documented recurrent depression with five near-fatal suicidal attempts, three of which had been in the past two years. The diagnosis was made using ICD–9 (World Health Organization, 1978) criteria. During his latest two-year admission he had, at intervals of six to eight weeks, episodic depressed moods with loss of appetite, anhedonia and low self-esteem. His suicidal thoughts were accompanied by frequent second-person auditory hallucinations – an unknown male voice immediately behind him telling him to hang himself – which occurred in clear consciousness. Computerised tomography, several electroencephalograms, and haematological profiles were normal. An extended severe depression led to urgent admission and six sessions of electroconvulsive therapy (ECT) that helped transiently.

Further depressive episodes recurred despite trials of imipramine, amitriptyline, phenelzine, chlorpromazine and lithium. Suspicion of psychomotor seizures from the patient's report of macropsia and headache concurrent with the hallucinations led to an unsuccessful trial of carbamazepine (200 mg daily). His medication at the time of psychological treatment was carbamazepine (200 mg daily; serum level 5.0 mg/l), lithium (400 mg nocte; serum level 0.9 mmol/l), chlorpromazine (200 mg daily), indomethacin 75 (4 mg) and paracetamol (p.r.n.) for headache.

On his own inspiration the patient bought a portable audiotape cassette and made a tape to play whenever he heard the voice (he had seen patients being treated for obsessive thoughts in such a manner). His initial tape was of his own voice shouting back at the hallucinated one but this did not help. He then decided instead to record and play pleasant memories of his family, work and holidays. He told staff that this tape reduced the duration of his hallucination and made him feel much better.

A case study was designed to test this impression. Each day was divided into alternating six-hour periods: A = switch the tape on if hallucinations come on; and B = don't use the tape at all (the earphones were worn during both periods when the voice occurred). The patient was asked to record the duration and intensity of auditory hallucinations during all periods. Statistical analysis of the results was done using Student's *t*-test. To check the validity of his records he was asked to produce them periodically and was questioned on their contents, and they were compared with nursing and medical observations.

Over 35 days the patients had 25 hallucinatory bouts in three clusters, each lasting up to three days. He used the tape in nine of the bouts and did not, in 14. Twice he used the tape in error during a B period when he should not have;

these results were included in the calculations for tape use (period A). His record keeping was good and consistent with nursing reports of his ward behaviour. The hallucinatory episodes lasted for an average of 4.4 min while listening to the audiotape and 22.2 min when not listening to it ($P<0.005$). When a bout began it took an average of 1.6 min for him to put on the headphones and start listening to the tape after which the hallucinations continued for a mean of 2.8 min. The mean time spent listening to the tape was 15.3 min per bout, of which a mean of 11 min followed the end of a bout. The tape greatly reduced the duration but not the frequency of the voice. Fifteen months later the patient was continuing to use his tape, saying that it was the only way to make the voice go away. Regular depressive periods continued until his death (myocardial infarct) 18 months after audiotape therapy had begun.

Discussion

During 35 days of audiotape therapy the hallucinatory bouts reduced markedly in duration but not in frequency, and nursing records reported less motor agitation and talk of suicidal intent. The patient reported by Morley (1987) also showed consistent improvement on self-report and behaviour.

Our patient had 40 years of recurrent depression which had become more severe with greater suicidal risk over the previous two years, requiring continuous day- or in-patient care. He had many treatments including ECT, three antidepressant drugs, phenothiazines and lithium in several combinations. These helped his depressions somewhat, but he remained extremely distressed by the voice commanding him to commit suicide. This drove him to seek a way of reducing the hallucinations. In a schizophrenic sample 13% would try to stop the auditory hallucinations on their own initiative, using stimulating music or loud noise and with a good or fair outcome (Falloon & Talbot, 1981). The present case study differs from previous studies both in the diagnosis (depressive disorder v. schizophrenia) and in the phenomenology of hallucinations (second person intermittent auditory v. third person continuous in most others). The decreased duration of the hallucinations in our patient resembles other findings of short-term improvement from listening to sounds that were meaningful for the patient (Margo *et al*, 1981). The latter study is unique in studying seven patients in nine experimental conditions, controlling for content and duration of auditory input. It concluded that external auditory stimulation was helpful if it had structure, meaning and demanded attention. Slade's (1976) idea that hallucinations are influenced by both internal and external stimulation is also supported by Feder's (1982) report of a patient who showed improvement by listening to a radio with a single ear plug; another patient was helped markedly by using an earplug in one ear but only helped marginally by music through headphones.

The previous studies and the present one underline the possibilities of treating auditory hallucinations by altering external stimulation; audiotape therapy may be helpful. Such a treatment needs to be investigated across a range of diagnoses.

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Delusional Misidentification of Familiar Inanimate Objects

A Rare Variant of Capgras Syndrome

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A 52-year-old woman presented exclusively with delusional misidentification of inanimate objects in the context of a short-lived acute psychotic episode. Certain theories are useful in understanding the formation of delusions of doubles in the patient (e.g. processes intrinsic to the depersonalisation/derealisation phenomena may have been aetiologically significant), but the actual choice of objects remains difficult to explain.

The syndrome of Capgras consists of the delusion that a familiar person or persons have been replaced by impostors (Capgras & Reboul-Lachaux, 1923). However, a number of cases of delusional misidentification of inanimate objects have been reported in the literature (e.g. Coleman, 1933; Kiriakos & Ananth, 1980; Anderson, 1988). We report here a case presenting solely with delusional misidentification of inanimate objects. We also briefly review the literature and discuss some of the explanatory theories.

Case report

Mrs S is a 55-year-old married woman who works full time as a packer in a components factory. Three weeks before admission to hospital she became argumentative and irritable, mainly with her husband. This had been preceded by some changes in her work-place involving a reduction in bonuses and a shift to less familiar work, both of which left her unhappy and angry. One week later she decided not to go to work and telephoned the management saying: "There is something strange going on in my head". By this time her irritability had become more pronounced and she began using uncharacteristically abusive language towards her husband. She also shut herself in the house and started showing bizarre behaviour by tearing up catalogues and magazines and arranging them into neat piles. She later went on to pack all her clothes into large bin bags ready to be disposed of, saying that none of the clothes were hers. At this time she expressed the belief that a number of familiar objects had been replaced by near-identical duplicates. These included almost all her clothes (with the exception of a nightdress which she continued to recognise as her own and wore continuously until admission), most of the crockery, their gas fire, washing machine, a certain picture in their living room and the road sign outside their front door.

After recovery, she was able to recall the exact circumstances surrounding her realisation that these objects had been changed: she was cleaning up one day at home when suddenly the world around her looked changed. She was disturbed about this and began looking closely at some of the objects which looked different. She then noticed marks, scratches and hair-line cracks which she believed were not there before. Her clothes looked too worn and either slightly shorter or longer than they should be. She also noticed that a picture of a tiger in their living room had whiskers, believing that the original did not. Her conclusion was that these items were not the originals, the latter having been mysteriously replaced.

A few days before admission, her eating became erratic and she shut herself up in her bedroom for most of the day. When she was visited by the psychiatrist (RTA) she refused to be interviewed and most of the talking was done across the firmly shut door with her replies consisting mainly of shouting and abusive language.

There was no history of alcohol or drug abuse, whether illicit or prescribed.

Admission was arranged under a section of the Mental Health Act and further examination confirmed that her belief that certain household objects had been replaced was held with total conviction. However, no explanatory delusions or elaborations were expressed on admission, or at any point during her illness.

Mrs S was clearly suspicious of staff, patients and relatives, although less so towards the latter. She was vaguely suspicious that her husband knew what was going on but this never developed into a paranoid delusion. She also appeared perplexed and had the feeling that there were things going on around her (both at hospital and at home) which she could not explain, a state approximating delusional mood. Her affect was mainly that of anger and no evidence of depression or elation could be found. Cognitive functions were intact on clinical testing and at no time during her stay in hospital did she show any disturbance in orientation or memory.

Her insight, however, was poor and on arrival to hospital she promptly appealed against her detention order. No evidence was found of delusions of doubles involving any of the significant people in her life (husband and two sons) or anyone else.

She has a past history of having suffered from three or four minor episodes of anxiety, agoraphobia and depression, none of which was severe enough to require psychiatric help. All of these episodes were self-limiting, not lasting more than a few weeks each time and usually related to some stressful life situation.

She remained isolated on the ward and initially refused to accept meals. The only reason she gave for this was that, as she did not accept being in hospital, it would be hypocritical of her to accept the food. As a result food had to be brought by her family.

Two weeks after admission, during which time she was treated with chlorpromazine orally, she showed a dramatic shift in her affect, becoming much more sociable and pleasant and at the same time beginning to express doubts about whether the household items had really been changed. A couple of days later she became totally symptom-free and managed a weekend at home with no problems. She was eventually discharged four weeks after admission on a small maintenance dose of chlorpromazine, which was discontinued three months after discharge. She remained well at six-months follow-up.

Physical investigations, including routine blood counts, urea and electrolytes, liver function tests, chest X-ray, thyroid function tests, electroencephalogram and computerised tomography, all yielded negative results.

The patient was tested on a number of psychological instruments. She had a full-scale IQ, measured on the Wechsler Adult Intelligence Schedule, of 104. Since this is marginally greater than the estimated pre-morbid level of between 95 and 100 (based on her interests, occupational history, and educational attainment), it is unlikely that there had been any deterioration in her level of intelligence at the time of testing. The subtest scatter and verbal/performance discrepancy were within normal range. Performance on the Wechsler Memory Scale resulted in a memory quotient of 107 and indicated good verbal and visual recall.

Performance on the Kendrick Battery indicated good digit copying speed and normal visual memory for objects (indicating good left temporoparietal function).

To assess finer visual functions, the visual function section of the Luria-Nebraska Neuropsychological Battery was administered, the results of which indicate visuospatial performance and spatial orientation abilities slightly above the normal population mean, and significantly higher than the cut-off point for brain damage, suggesting no right-hemisphere dysfunction. Her scores on the Personality Assessment Schedule (Tyrer & Alexander, 1985), used to identify a possible paranoid personality trait, showed no evidence of personality disorder of any type.

Therefore, the results of both physical and psychological investigations showed no evidence of organic brain dysfunction at the time of our examination.

Discussion

The patient presented with a number of unusual features. The delusional misidentification of inanimate objects was not accompanied by any other delusions and this occurred within the context of a short-lived psychotic episode. The majority of reported cases of the Capgras syndrome have been diagnosed as suffering from schizophrenia or affective disorder, with a smaller number occurring in patients with organic brain disease (Kimura, 1986). None of these diagnoses was evident in our patient. Other cases of delusional misidentification of inanimate objects have been reported in the literature. However, with very few exceptions (e.g. Anderson, 1988), most have either been accompanied by the delusional misidentification of persons (Enoch, 1986), or the inanimate object represented a clear substitute for the person (e.g. personal letters; Coleman, 1933). We also believe that this case is the first to be reported with a Capgras syndrome precipitated by a psychosis which appears to be psychogenic.

According to psychodynamic theory the delusion of doubles is a solution to the problem of ambivalence through the mechanisms of splitting and projection (Enoch & Trethowan, 1980; Berson, 1983). Our patient had delusions of doubles involving a relatively large number of objects. Some items, such as the road sign and gas fire, were of no particular emotional significance according to the patient's own self-report. Although we accept that individuals may have ambivalent feelings towards inanimate objects of high emotional value, this explanation seems unlikely in the case of some of the objects reported here.

The depersonalisation/derealisation phenomena has been noted by a number of authors in patients with the Capgras syndrome (Christodoulou, 1986*a*). Mrs S certainly experienced intense feelings of unreality at the time of the onset of her symptoms and for several days afterwards. The process of disengagement and feelings of lack of familiarity, intrinsic to the depersonalisation/derealisation phenomena, may have been aetiologically significant in the formation of delusions of doubles in our patient. It is conceivable, in such a state, that the thought that mysterious substitution of a person or object has taken place can arise if this occurs within the context of a paranoid psychotic state.

Fascination with the concept of doubles is evident in many cultures, and is illustrated by the inclusion of the concept within mythology and literature (Christodoulou, 1986*b*). This interest, which has transgressed cultural barriers, suggests that the concept of doubles may represent an archaic form of thinking which can be reactivated under certain circumstances.

The choice of object(s), however, remains difficult to explain and this area will, no doubt, continue to attract considerable interest and speculation.

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Capgras Syndrome in a Patient with Migraine

M. S. BHATIA

Capgras syndrome in a patient with migraine is reported. It is difficult to establish whether this association, which has only been reported once before, had aetiological significance or was coincidental. Investigating all cases of Capgras syndrome for organic pathology by MRI is suggested.

Capgras syndrome is considered a rare entity in which the patient believes that a person, usually a close relative, has been replaced by an exact double or impostor and maintains this false belief despite evidence to the contrary (Capgras & Reboul-Lachaux, 1923). The syndrome has usually been described as a manifestation of schizophrenia, although it has also been reported as occurring in association with affective disorders (Signer & Isbister, 1987). Recently, there have been many case reports describing its occurrence in association with such conditions as brain tumour (Todd *et al*, 1981), dementia (Kumar, 1987), diabetes (MacCallum, 1973), reactive hypoglycaemia (Bowerman, 1981), hypothyroidism (Madakursira & Hall, 1981), viral encephalitis (Nikolovski & Fernandez, 1978), pneumonia (MacCallum, 1973), hepatic encephalopathy (Pies, 1982), chloroquine toxicity (Bhatia *et al*, 1988), folic-acid deficiency (MacCallum, 1973), and vitamin-B12 deficiency (Zucker *et al*, 1981).

The following report discusses the presence of the Capgras syndrome in a patient with migraine.

Case report

The patient was a 35-year-old married woman from a nuclear family of lower socioeconomic status. There was no family history of psychiatric disorder or epilepsy. Her birth and early development were uneventful. She was brought to the out-patient department by her husband who complained that she had been talking irrelevantly and had had an excruciating headache of sudden onset for about 12 hours. The headache had started on the right side and then became generalised. It was associated with vomiting, sweating, photophobia, watering and redness in both the eyes. The patient's migraine attacks, which lasted for 48 hours, occurred once a month. There was neither any identified precipitant nor any recent change in the frequency of her headaches. Her 'irrelevant talks' included saying that her husband was dead and the actual person was an impostor. During this episode her husband tried to persuade her of his true identity by showing her photographs and reminding her of their previous life but without success.

There was no deterioration in her personal appearance and appearance and hygiene and no disturbance of psychomotor activity. Her affect was appropriate and she was not hallucinating. Her orientation and memory were intact. There was no evidence of formal thought-disorder, depression or elation. Physical and neurological examinations revealed no abnormality. Blood biochemistry as well as computerised tomography was normal. An electroencephalogram showed bilateral theta-waves over the temporal regions. On the Wechsler Adult Intelligence Scale her full-scale IQ was 110.

She was prescribed aspirin (2 g daily) and haloperidol (15 mg daily) in divided doses. Her headache subsided in a day but the Capgras delusion took three weeks to disappear. The dose of aspirin was reduced to 1 g daily while haloperidol was gradually tapered off over six weeks. During follow-up for six months, there was no recurrence of any of these symptoms including the Capgras delusion.

Discussion

Although reports of Capgras delusion co-existing with various brain lesions and systemic disorders are appearing with an increasing frequency, the association of Capgras syndrome with migraine has only been described once before in the literature (MacCallum, 1973). That case had a right-sided hemiplegia secondary to basilar migraine, but the present case showed no neurological deficit. Subjects with migraine have been found to show an abnormally high incidence of generalised asynchronous slow-waves and other abnormalities between the attacks (MacCallum, 1973). The evaluation of bilateral theta-waves is exceedingly difficult, and these may be seen in deep-seated midline cerebral lesions, generalised convulsive disorder or even in normal adults (MacCallum, 1973; Christodoulou & Malliara-Loulakaki, 1981).

It is difficult to establish whether the Capgras delusion is caused by the presumably organic pathology that produced the migraine or the psychogenic process arising from the pain and other symptoms of the migraine, or whether the simultaneous occurrence of both migraine and Capgras syndrome was coincidental. However, it would seem desirable to investigate all cases for an organic pathology by magnetic resonance imaging (MRI), since, in the case of a patient who developed Capgras syndrome as part of an interictal psychosis, MRI revealed bilateral subcortical lesions in occipitotemporal and frontal regions (Lewis, 1970).

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Unwanted Erections in Obsessive–Compulsive Disorder

HILARY M. C. WARWICK and PAUL M. SALKOVSKIS

The presentation of inappropriate penile erections calls for great caution in making a diagnosis. Two patients who had unwanted erections also had obsessive–compulsive disorder – a previously unreported association. Obsessive–compulsive disorder is *not* inconsistent with unwanted erections and should be considered in assessment.

Obsessive–compulsive disorder has a diverse content; it is estimated that 5–9% of obsessional patients experience intrusions of a sexual nature (Aktar *et al*, 1975; Stern & Cobb, 1978). Such obsessions are often particularly repugnant and distressing to patients, who may find it hard to admit to their worries (Salkovskis & Kirk, 1989).

In a further group of obsessional patients, sexual activity may be avoided as a consequence of intrusive thoughts about contamination. Behavioural treatments are successful in up to 85% of obsessive-compulsives (Foa & Goldstein, 1978), and there is no data to suggest that patients troubled by sexual obsessions respond any differently.

Although there is evidence that anxiety can facilitate sexual arousal (Bancroft, 1974; Barlow *et al*, 1983), the occurrence of unwanted erections in obsessive–compulsive disorder has not previously been recognised. There are considerable difficulties in diagnosis and management of such presentations, particularly in the absence of previous reports. These difficulties can only be clarified by a careful examination of the clinical features and psychological processes involved in each case. We report two cases of obsessive–compulsive disorder where unwanted erections were described in association with obsessional phenomena to the great distress of the patients. To our knowledge, this is the first published report of this phenomenon.

Case one

A 43-year-old married man with an 18-month-old daughter had been successfully treated for obsessive–compulsive disorder using cognitive-behaviour therapy. He experienced a wide range of intrusive thoughts and imagery, associated with extensive overt and covert neutralisation. The central theme was always that he might be responsible for harm befalling his wife and child and had to try to prevent this by ritualising. Towards the end of this treatment he admitted for the first time that he was frequently experiencing erections when he was with his daughter. Erection could occur immediately before, immediately after or simultaneously with anxiety-provoking intrusive thoughts and images of a sexual nature. Erections, intrusive thoughts and images were perceived as alarming, repugnant and unwanted and were strongly resisted. He neutralised these intrusions by producing 'good' thoughts and images and began to avoid the child as much as possible. When he had to be alone with the child, he immediately tried to suppress erection and thoughts. He interpreted the combination of intrusive thoughts and erections in a particularly negative way; for example: "This must mean I am a pervert", "This means I might assault my daughter". Unlike his other obsessions, he was unable to confide in his wife, and he became increasingly depressed with occasional suicidal thoughts. He had at *no time* had, and had *no* desire to have, sexual contact with the child.

Once the diagnosis had been clearly established, then treatment was instigated. Initially, this involved instructions to stop avoiding his child, a programme of graded exposure and instructions in response prevention (focused on his covert neutralising; see Salkovskis & Warwick, 1988). Although he had previously used exposure and response prevention successfully, he felt unable to comply because of extreme anxiety levels. Careful enquiry revealed that he felt that the occurrence of unwanted erections made this 'different' from his other obsessions and was afraid that the feared consequence might occur.

Assessment revealed that he believed that he should always be able to control and prevent unwanted erections; his failure to do so made him think that he might be a pervert. Patient and therapist then discussed an alternative explanation which could be readily applied to his problem. Blushing was used as an example of a common autonomic response which cannot always be controlled and which occurs in situations associated with anxiety. Furthermore, blushing tends to occur paradoxically when suppression is attempted. The common role of blood flow changes was a further parallel. After this discussion, the unwanted erections virtually disappeared and the patient resumed normal contact with his child without ritualising. Twelve months later he remained symptom free.

Case two

The patient was a 38-year-old polytechnic student. He presented at the emergency department of the general hospital, complaining of 'evil thoughts' and requesting psychiatric admission. Four years previously, he had been admitted to a psychiatric hospital with depression and intrusive thoughts; he made several suicidal attempts at this point. He had previously been diagnosed erroneously as schizophrenic, apparently on the basis of his obsessional thoughts and a six-hour psychotic episode precipitated by amphetamines. Although it was evident that he had been troubled by intrusive thoughts for several years, the present episode had begun three days previously when a colleague was visited by someone with children. The patient had thoughts concerning sexual and violent assault, and had become increasingly anxious about the possibility that he might act on these.

When he was first seen, he was extremely reluctant to describe his thoughts, and the details emerged over the course of several sessions. Previous thoughts had concerned sexual and violent acts towards women. During the episode leading to presentation, the thoughts centred on violent sexual assault of children, although within two weeks this had extended to women and to men. He found these thoughts extremely repugnant and said that he definitely did not want them. He was quite clear that the intrusions were the product of his own mind, but found this fact to be very alarming. His reaction was to try to suppress the thoughts, to avoid situations where he might encounter children and to repeat stereotyped phrases to himself when the thoughts occurred, such as "I won't do it"; "It's prison for me". He constantly sought reassurance about how likely it was that he might act on his thoughts. He would verbally check whether he might have accidentally assaulted someone. During therapy sessions he would talk very quietly and frequently check that he could not be overheard. A major factor in his fears was the fact that his brother had, four years previously, been convicted of assaulting two women. The patient had interpreted his own intrusive thoughts as a sign that he might act in the same way.

Two months after his first attendance, he described two different types of circumstances where erections were associated with his obsessional thoughts. Firstly, he would experience intrusive images (always concerning children) when he masturbated to photographs of adult women. Initially, he had tried to suppress these images; when they continued to occur, he tried to desist from masturbation altogether. He was concerned that the occurrence of intrusions at this time indicated a possible subsequent loss of control and 'acting out'. Secondly, he noticed that he had an erection on at least two separate occasions when intrusive thoughts and images of violence to children occurred in the context of standing near children during a treatment programme. He found this second experience particularly alarming, because it seemed to him to be a sign that he was on the verge of losing control and attacking the children he was near. He reported that this fear was based on the thought "I have already kind of lost control; my body is out of control". In both instances, he found the erections extremely repugnant. Treatment was prematurely terminated as the consequence of a major life event which resulted in the patient moving to another part of the country.

Discussion

It is vital to establish the diagnosis where penile erection is reported in association with unusual stimuli. In these cases, the erections and the associated intrusive thoughts and images were regarded as repugnant and senseless. They were strongly resisted and were associated with avoidance and overt and covert ritualising. There was no urge to have sexual contact with children and the patients continued to have normal sexual fantasies and/or relationships. There was no history of paedophilia and sexual fantasies involved only adult women. The first patient's obsessions had previously concerned topical issues (e.g. hepatitis B and AIDS), and the unwanted erections coincided with publicity about child abuse. In the second patient, his brother's previous conviction for assault played an important role in his concerns. These factors confirmed the diagnosis of obsessive–compulsive disorder, and paedophilia was excluded.

This phenomenon could be an example of discordance between autonomic sensations and demonstrable autonomic changes. However, both cases *said* that their erection was palpable and it was visible to the one patient in whom circumstances allowed inspection. While this report would have benefited from objective measurement of erections, this was considered unjustifiable in these clinical cases, who were not involved in a research study. Both were alarmed by the experience and perceived implications of unwanted erections, and objective measurement would have clearly added to their distress, without any clinical benefit. Future studies of this phenomenon might attempt such measures, before and after treatment. In these cases, the perception of erection was the source of alarm. Conclusions here necessarily concern *perceived* sexual arousal, although this correlates well with actual changes in penile circumference (Bancroft, 1974).

Erections were associated with obsessional intrusions in two ways: (a) obsessional intrusions occurring during usual sexual activity; and (b) intrusions directly associated with erections. In the former unwanted intrusive thoughts and images occur during sexual arousal *unrelated* to the intrusions. Thus, case two reported that masturbation to his usual heterosexual stimuli was interrupted by obsessional intrusions. Attempts to suppress intrusions tended to increase their frequency and led the patient to refrain from masturbation. Barlow (1986) reports that sexual dysfunction is often associated with distraction by irrelevant (non-erotic) stimuli and a paradoxical effect has been shown to operate when attempts are made to suppress intrusive stimuli (Salkovskis, 1989). These phenomena may account for the impairment of sexual relationships in some obsessional patients.

Given the conventional view that sexual arousal and anxiety are incompatible, the association between erections and obsessional intrusions which provoke anxiety is unexpected. However, there is evidence that the relationship between sexual arousal and anxiety is more complex. In studies on aversion therapy in homosexuals, Bancroft (1974, p. 126) observed that *increased* sexual arousal sometimes followed aversive (shock) stimuli, possibly because of contrast effects. More recently, Barlow *et al* (1983) reported that, in men with no history of sexual dysfunction, anxiety related to the threat of shock *enhanced* sexual arousal as indexed by penile plethsymography. This effect was slightly increased if the shock was perceived by the subjects as being contingent upon the subjects' erectile response, i.e. if the erectile response were itself the focus of anxiety. These data suggest that, for obsessionals in whom there is some association between sexual stimuli and anxiety-provoking thoughts, the occurrence of sexual arousal is a not unlikely consequence. If a patient with an obsessional disorder reports associated erections, the diagnosis of obsessive-compulsive disorder is not automatically called into doubt.

When apparently inappropriate sexual arousal is detected in the context of an obsessional problem, it is important that a diagnosis is reached on the basis of the phenomenology of the problem as a whole rather than the occurrence of isolated symptoms. It is obviously vital that sexual deviation is accurately excluded. The association of obsessional thoughts and sexual arousal may occur relatively frequently, but seldom be revealed because of associated guilt and fear of the consequences of disclosure. An inexperienced therapist who is unaware of the possibility of an association between obsessional problems and sexual arousal may fail to implement proper behavioural treatment because of doubts about the nature of the phenomenon, and perhaps also communicate these doubts to the patient. Treatment of obsessive-compulsive disorder involves the explanation that the occurrence of obsessions represents the intrusion of unwanted and senseless thoughts into consciousness. Acceptance of this view is important for compliance with behavioural treatment (Marks, 1987), but both our patients found the occurrence of erections an obstacle to their acceptance of the treatment rationale. Patients have found the following explanation particularly helpful and easy to understand: "Erections are a reflexive response involving changes in blood flow, and can be regarded as similar to excessive blushing in social situations. As often happens with blushing, the more anxious one becomes about the response itself, the more likely it is to happen, because the reflex can be paradoxically set off by anxiety." Careful explanation about the nature of unwanted erections is crucial – without this appropriate behavioural treatment may be unacceptable to these patients.

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Aerosol Addiction

V. P. PRASHER and J. A. CORBETT

A case of dependency on prescribed pressurised aerosols in a patient with asthma and mild mental handicap is reported. The majority of reported cases involve young asthmatics, abuse being reported mainly using salbutamol inhalers although other inhalers have also given cause for concern. The agent of addiction is uncertain although it may be the fluorinated hydrocarbons used as propellants, rather than the active substance itself.

Drug abuse using the lungs as the source of entry is nothing new, having initially involved smoking of cannabis and opium. In recent times, however, abuse of solvents such as toluene and acetone has caused concern. Abuse of prescribed aerosols (sympathomimetic and steroid inhalers) is largely unknown although periodic case reports have been published (Table I). The majority of cases reported relate to mentally normal young people with asthma. The authors report a young asthmatic adult with mild mental handicap who abused his inhalers more frequently than in previously described cases.

TABLE I

Review of reported cases of aerosol addiction/abuse

Author(s) & year	*Age: years*	*Substance abused*
Edwards & Holgate (1974)	24	Salbutamol inhaler
Gluckman (1974)	51	Salbutamol tablets inhaler
Gaultier *et al* (1976)	22	Salbutamol inhaler
Kjelleman (1977)	14	Salbutamol respiratory solution (administered by PARI machine)
Pratt (1982)	15	Salbutamol inhaler
	15	Salbutamol inhaler
Thompson *et al* (1983)	17	Salbutamol & beclomethasone inhalers
Brennan (1983)	13	Salbutamol inhaler
	15	Salbutamol inhaler
	12	Salbutamol inhaler
	12	Salbutamol inhaler
	11	Salbutamol inhaler
Wikramoshinghe & Liebeschnetz (1983)	11	Salbutamol inhaler
Raine (1984)	17	Salbutamol inhaler
O'Callaghan & Milner (1988)	4	Salbutamol & beclomethasone inhalers

Case report

A 27-year-old man of Sikh extraction with mild mental handicap and asthma was admitted to hospital with aggressive behaviour. Born in India following a normal pregnancy and delivery, his early developmental milestones were also normal. He came to England with his parents at the age of six. At school he was noticed to be slow in reading and writing (even allowing for cultural differences) and was diagnosed as having mild intellectual impairment of unknown origin by the school psychologist. He remained physically well and mentally stable until a visit to India at the age of 19 years during which he underwent an arranged marriage.

Two months later he had his first severe asthmatic attack. There was a family history, with his father also being asthmatic. He made a good recovery following treatment with a salbutamol inhaler. Over the subsequent years, further attacks were again treated with inhalers, containing either salbutamol and/or terbutaline sulphate. However, during this period, abuse of the inhalers occurred. Initially, he used two or three a week but this increased up to a maximum of six to eight a week. Consumption of the inhalers occurred as if they were 'confectionery'. The family noticed a temporal relationship between the inhaler abuse and aggression. While using the inhaler no aggression was seen. However, once it was finished he would demand another and if this was not available, both verbal and physical aggression occurred.

In 1984 (aged 22 years) admission to hospital occurred under Section 3 of the Mental Health Act 1973. He presented with continuous substance abuse and related aggression. There was evidence of a psychosis, with bizarre behaviour involving depositing milk bottles in nearby streets, delusions of grandeur where he felt he could cure all his family ailments, repeated clicking of his fingers, and reduced insight. Management involved reduction in aerosol use, substituted with theophylline and addition of chlorpromazine (50 mg t.d.s.). All symptoms settled within a few days with no evidence of aggression or psychosis on discharge.

During the following year, further asthmatic attacks occurred and with reintroduction of the salbutamol inhaler abuse again followed. He was readmitted because of further verbal and physical aggression. Monitoring of inhaler use led to elimination of aggressive behaviour, with discharge 72 hours later.

The most recent admission occurred because of unacceptable aggression while demanding his terbutaline inhaler. The patient had punched and kicked his sister, thrown a cup across the room at his brother, permanently damaging his eye and pulled his father's beard while demanding his inhaler. He had also poured hot water over another sister and at one point, threatened his family with a kitchen knife. Violence was not confined to the home. On several occasions he caused a disturbance at both his general practitioner's surgery and local pharmacy leading to the recipient giving way and supplying further inhalers.

On this admission, no psychotic symptoms were demonstrated. His inhalation treatment was stopped completely and theophylline (200 mg b.d.) started which controlled his asthma. Once again, the patient settled quickly within a few days, being discharged home on oral bronchodilator medication, no abuse or aggression occurring to date. Other substance abuse (including alcohol, nicotine and caffeine) was absent throughout.

Discussion

Pressurised aerosol treatment for bronchospasm has revolutionised the management of asthma since its introduction 20 years ago. Generally, it is a safe and effective form of treatment.

Since Gluckman (1974) brought to attention the possible abuse of such treatments, few cases have been reported in 15 years (Table I). The majority are teenagers or young adults although exceptions of a child aged three and a half years and a man aged 51 years have been reported. All cases are of known asthmatics although as Brennan (1983) points out, in the young group, non-asthmatics can also become involved in such addiction. The present case report illustrates that people with mental handicap may be vulnerable to such behaviour.

The question whether this activity of deliberate repeated excessive consumption of aerosol substance is true dependence or not needs to be scrutinised closely. Dependence syndrome has recently been defined as including a strong desire or compulsion to use a substance, often referred to as 'craving'. There is evidence of impaired capacity to control initial use and levels or to terminate use. It may also include use of the substance to avoid withdrawal symptoms, evidence of tolerance, narrowing of patterns of substance abuse, progressive neglect of alternative pleasures, behaviours or interests, and persistence with substance use despite clear evidence of harmful consequence (World Health Organization, 1989).

Certainly the case report fulfils this definition as do many of the reviewed cases.

A further question of paramount importance which needs to be answered is, to which agent in the inhaler does addiction occur? The active antispasmodic in all previously reported cases was salbutamol (beclomethasone additionally in two cases, Table I). However, now terbutaline has also been incriminated.

Salbutamol has been reported to have antidepressant properties and terbutaline (another B2 adrenergic receptor stimulant) may act similarly (Widloche *et al*, 1977). However, studies using radiolabelled salbutamol in rats resulted in virtually none being distributed in the brain (Martin *et al*, 1971). Addiction to fluorocarbons used in domestic aerosols results in euphoria and intoxication (Bass, 1970). It has been shown that fluorocarbons are absorbed into the blood after inhalation (Dollery *et al*, 1970).

On balance, it is most possible that fluorinated hydrocarbons, rather than the active agent, are responsible for the stimulating and subsequent addiction effects. If this is the case, conversion to a dry powder system such as Rotacaps and Rotahalers or Diskhaler is recommended. This also allows for more accurate dose monitoring.

Pressurised aerosols are widely used with unquestionable benefit to the majority of asthmatic individuals. Abuse by a few must not, therefore, create disproportionate fear of addiction in vulnerable people in today's society of increased drug awareness. However, awareness of the possibility of aerosol addiction must exist among those in the caring professions.

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British Journal of Psychiatry (1990), **157**, 924–928

Three Thousand Days of Pregnancy

A Case of Monosymptomatic Delusional Pseudocyesis Responding to Pimozide

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An unusual case of pseudocyesis of almost ten-year duration, characterised by a monosymptomatic hypochondriacal delusion and a selective response to pimozide is reported. The nosological status of the diagnosis, delusional (paranoid) disorder, is suggested to be a variety of the Kraepelinian concept of paranoia. The therapeutic pitfalls of relying on a phenomenological distinction between overvalued ideas and delusional beliefs are discussed.

Hypochondriacal or somatic delusions often occur in the course of major depression and schizophrenia (Munro, 1982). Where the clinical picture is dominated by a solitary hypochondriacal delusion, not secondary to another psychiatric disorder, the condition is known as monosymptomatic hypochondriacal psychosis (MHP). Despite a wide individual variety in delusional content, the clinical presentations are very similar and MHP is essentially synonymous with the Kraepelinian concept of paranoia: a chronic, encapsulated, monodelusional disorder in which the personality is relatively unaffected (Munro, 1982). Whereas Kraepelin himself abstained from delimitating a hypochondriacal form of paranoia, DSM-III-R (American Psychiatric Association, 1980) has recognised the concept of MHP under the rubric of delusional (paranoid) disorder, somatic type.

Pseudocyesis (literally 'false pregnancy') is an uncommon condition, characterised by a conviction of pregnancy, often although not invariably associated with the somatic manifestations of the gravid state (Cohen, 1982; Trethowan, 1979). It is not restricted to women (Evans & Seely, 1984), can occur before or after the menarche or menopause (Barglow & Brown, 1972), and has been described under a variety of psychiatric nosological categories, including somatoform and mood disorders and schizophrenia (e.g. Mortimer & Banbery, 1988). The belief can range from a fleeting idea to a tenaciously held conviction (Cohen, 1982). Distinguishing between psychotic and non-psychotic varieties of pseudo-cyesis, simulated pregnancy, and pseudopregnancy caused by tumours, or endocrine changes mimicking symptoms of gravidity, may be difficult because elements of each diagnosis are often intermingled (Miller & Maricle, 1988).

According to Trethowan (1979), the somatoform variety of pseudocyesis, ostensibly more common in younger patients, has a psychotic analogue, akin to delusional hypochondriasis, which usually occurs in childless, often single, menopausal women. Some cases of pseudocyesis may represent a form of MHP (Bjerg Hansen, 1976; Cohen, 1982). However, a survey of the Anglophone literature revealed only two reports suggestive of monodelusional pseudocyesis in *women* and, from the information available, the correct diagnoses are not entirely clear (Barglow & Brown, 1972; Reichenbacher & Yates, 1987). A more convincing *female* case, with a nine-year history of intermittent delusions of gravidity, was reported from Czechoslovakia (Vacek, 1980). Finally, Neppe (1989) has published an account of a Xhosa *man* from South Africa who developed an isolated delusion of pregnancy after a homosexual encounter with a witch doctor.

Case report

Ms U is a 35-year-old unemployed woman, born in the UK of Polish parents. Her early childhood was uneventful but she retained vivid memories of her mother's traumatic ectopic pregnancy 15 months before the birth of her sister who was ten years younger than her. She left school at the age of 16 and, after completing a two-year secretarial course, worked for a number of years as a typist and receptionist. Premorbidly lively and outgoing, she enjoyed being the centre of attention but was generous and had regard for others. There was no family history of mental disorder.

She was unprepared for her menarche at age 11 years, and her periods were heavy and often painful. When she was 14 a film about childbirth left her with feelings of fear and revulsion about sex, birth and babies. Shortly afterwards she was raped by a youth. Although terrified of becoming pregnant, she did not confide in anyone for fear of reproach. She had a boyfriend at 18 years but did not enjoy intercourse as it reminded her of the assault.

In February 1978, aged 22 years, Ms U met her future husband, a 27-year-old semiskilled electrician from Poland. A two-month courtship culminated in a civil marriage which was consummated three months later after the traditional church wedding. Her husband was sexually naïve and she suffered from vaginismus, dyspareunia and anorgasmia. When she had to stop her oral contraceptive because of severe side-effects, her husband refused to use a sheath because he wanted to start a family. After two months of unprotected intercourse she began to 'feel something' in her stomach, complained of increasing abdominal pain, low mood and lack of energy, and lost 10 kg.

In January 1979, Ms U was diagnosed as suffering from "reactive depression with tremendously high anxiety levels". Described as "overprotected and spoilt", her problems were attributed to "the change to the responsibilities of being a married woman". She was prescribed phenelzine, trifluoperazine and diazepam without further follow-up. Six months later she was admitted to hospital for the first time. While her mood had improved, her abdominal pain had become worse and she was preoccupied with a fear of being pregnant, despite regular menses, the use of contraception by her husband and the negative results of several pregnancy tests. Her psychiatrist considered her "a self-regarding, acting-out young woman", and discharged her after a month to weekly dynamic psychotherapy. The therapist related her pregnancy beliefs to "her close-knit Polish family, her relationship with her father, her unhappy marriage and her desire to manipulate the men in her life and draw her mother and sister closer to her to avoid facing up to the real issues and conflicts". Her symptoms remained unchanged and she terminated therapy after three months. A referral to a therapeutic community was unsuccessful.

During 1980 and 1981 Ms U was seen as an out-patient and at one stage was thought to suffer from "mild anorexia nervosa" with "an overvalued idea of pregnancy which had become delusional". Treatment with pimozide was suggested but not implemented. Her original psychiatrist, however, continued to regard her as 'not mentally ill'. Her husband found her vociferous complaints of abdominal pain, demands for an abortion and suicidal threats increasingly difficult to tolerate and he finally abandoned her.

In April 1982 Ms U was admitted under a different psychiatrist who described her as a "labile histrionic person, subject to extreme anxiety, with an overvalued idea about pregnancy in a setting of a monosymptomatic (abdominal pain) hypochondriasis". Within six days of starting pimozide (8 mg/day) her pain and distress abated and she stopped voicing her conviction of pregnancy. Her parents and staff reported a dramatic improvement. For a few days she exhibited mild euphoria and disinhibition with pressure of speech. Pimozide was increased to 16 mg/day and she was discharged, fully recovered, after only a month. She eventually found work in a factory and over the next 17 months pimozide was gradually reduced. In November 1983, six weeks after its discontinuation, Ms U reported a return of abdominal pain which responded within three days when pimozide (4 mg/day) was reintroduced. Soon afterwards she had an unprotected sexual encounter and within a month all her previous symptoms returned.

An increase in pimozide (8 mg/day) brought a partial improvement but her compliance was suspect and in January 1984 her father applied for her compulsory admission to hospital. Gynaecological examination revealed a non-tender, left-sided ovarian cyst (4.5 cm in diameter) but it was not considered of aetiological significance. A pregnancy test was negative, and haematology, biochemistry and computerised tomography were unremarkable. She did not improve after a month on a combination of pimozide (24 mg/day), haloperidol (60–120 mg/day) and chlorpromazine (100–300 mg/day). Her scores on the Beck Depression Inventory fluctuated between 30 and 40, but ten sessions of bilateral electroconvulsive therapy, several months treatment with high doses of dothiepin, lithium, carbamazepine, diazepam and zuclopenthixol decanoate produced no amelioration of her beliefs or disturbed conduct. A behaviour-modification programme was similarly ineffective. Ms U repeatedly absconded and presented in casualty, general practitioner (GP) surgeries or chemists, demanding an abortion. In addition to abdominal pain, she also suffered from amenorrhoea and painful breasts.

Her continuous screaming caused considerable distress on the ward. Abreaction with methylamphetamine and methohexitone revealed nothing new except a fear of sex. A reintroduction of pimozide (12 mg/day), five months after its discontinuation, produced a response within two days, followed by a complete relapse a fortnight later after she had had unprotected intercourse. Despite taking an increased dose (16 mg/day) for another 12 days her symptoms remained unchanged and, after nine months in hospital, she was discharged on zuclopenthixol decanoate (400 mg/week).

During the following year Ms U attended her GP almost daily and bothered the neighbours, police and ambulance service, often screaming for hours. Despite having had an intra-uterine device fitted she still believed she was pregnant. Her psychiatrist suggested a leucotomy as a final therapeutic option but refused to readmit her. Following desperate pleas from her parents, Ms U's care was taken over by yet another team, and in November 1985, aged 30 years, she was admitted for the fourth time. Zuclopenthixol was discontinued and she began to menstruate for the first time in two years. The removal of her ovarian cyst left her mental state unchanged. A behaviour modification programme resulted in a severe deterioration of her mental state. Chlorpromazine (800 mg/day) and diazepam (15 mg/day) produced a modest improvement in her behaviour but she developed galactorrhoea and continued to lift her skirt to check if she was pregnant. Her behaviour programme was discontinued after ten months. A subsequent nine-month trial of sulpiride (400 mg/day) brought no relief and she took an overdose of weedkiller and paracetamol. The regional secure unit refused to admit her for a structured behavioural programme in view of her 'chronic hysterical condition'; the therapeutic community again rejected her as "a borderline patient who presented a phenomenon of insatiable greed". In August 1988, after a five-month drug holiday, pimozide was reintroduced. The dose was increased to 40 mg/day over four weeks, leading to a full recovery, again followed by a brief spell of disinhibition and pressure of speech. There were no signs of affective blunting or personality deterioration and her self-care and presentation improved dramatically. Accepting that she had never been pregnant, Ms U put her abdominal sensations down to anxiety. She began to plan a return to a secretarial career and was discharged to her own flat. At two-year follow-up she has remained well and, apart from mild galactorrhoea and akathisia, showed no adverse side-effects of her medication.

Discussion

Authors and clinicians frequently fail to distinguish between monodelusional hypochondriasis and superficially similar disorders of a non-psychotic type (Munro, 1982). The truly protean nature of this condition is illustrated by the plethora of alternative diagnoses which Ms U attracted during her decade of contact with psychiatric services. Nevertheless, examination of her records reveals several of the symptoms and characteristics identified by Munro (1982), on the basis of a series of 50 cases, as indicative of MHP. The fundamental feature of her presentation was a single, hypochondriacal belief of being pregnant, not secondary to any other psychiatric disorder. Although the delusion was relatively encapsulated, the effects of her distress were widespread. Her symptoms fluctuated in severity but never vanished spontaneously. Treatment with pimozide effected a singular and profound remission of her abnormal belief followed by a rapid resolution of anxiety, anger and secondary depressive symptoms.

Barglow & Brown (1972) conceptualised pseudocyesis as psychological or somatic manifestations which appear to originate from two interconnected components: an awareness of recent bodily change or disturbance, linked to emotional aspects of pregnancy. Delusional hypochondriasis may originate as a result of coenaesthopathy, the disordered experience of exaggerated or distorted proprioceptive, kinaesthetic or haptic sensations of which the subject is not normally aware, and a subsequent breakdown in the ability to distinguish between normal and abnormal somatic perceptions (see Munro, 1982). Similarly, minor, coincidental, bodily changes may initiate the false belief of pregnancy in a susceptible individual (Mortimer & Banbery, 1988; Reichenbacher & Yates, 1987). In psychotic patients prolactin secretion and galactorrhoea after neuroleptic treatment (Cramer, 1971) or ileus and intestinal dilation due to the anticholinergic side-effects of antidepressive and neuroleptic drugs (Evans & Seely, 1984) may stimulate, confirm and reinforce the desire, fear or belief of being pregnant.

The intensity of hypochondriacal concerns can range from an obsessional or overvalued idea to one that is frankly delusional. Such phenomenological distinctions can be difficult to delineate in clinical practice and the differences may only be one of degree (Opjordsmoen & Retterstøl, 1987). Indeed, descriptions of the development of delusional pseudocyesis (Barglow & Brown, 1972; Mortimer & Banbery, 1988; Neppe, 1989) support Kraepelin's concept of paranoia as the result of the interaction between personality factors and life experiences, the clinical phenomena arising to some extent understandably from defects in judgement. In the absence of other psychotic symptoms, the distinction between delusional and non-delusional forms of hypochondriasis may be problematic and in younger patients such judgements may be further compounded by the disruptive effects of psychological disturbances upon personality development. Since the therapeutic implications are important, a careful differential diagnosis is essential but in difficult cases a trial with a neuroleptic or antidepressive drug may sometimes be indicated.

The most consistent prospect of symptomatic relief in MHP has been offered by neuroleptics in general and the diphenylbutylpiperidine derivative, pimozide, in particular. Several factors may account for this. Pimozide's effectiveness as a dopamine receptor blocker and the limited psychopathology of MHP may indicate a relatively circumscribed underlying pathophysiology (Munro, 1982). Johnson & Anton (1983) relate its efficacy in delusions of infestation to pimozide's potency as an opiate antagonist. Drugs which block opiate receptors can act as central suppressors of abnormal peripheral sensations, stimuli which could precipitate, reinforce or maintain the delusional beliefs of some patients with MHP (Matas & Robinson, 1988).

Until the first reports of pimozide's usefulness in the treatment of MHP, the various forms of delusional disorder were regarded as largely persistent and refractory in nature, even if a deterioration to schizophrenia did not occur (Bjerg Hansen, 1976; Opjordsmoen, 1988). A substantial number of patients with hypochondriacal delusions have marked depressive symptoms and some eventually commit suicide (Munro, 1982; Opjordsmoen & Retterstøl, 1987. There are serious difficulties in engaging such patients in dynamic psychotherapy (Bjerg Hansen, 1976). Behavioural methods may alleviate some of the associated distress and abnormal behaviour but have little success in abolishing the delusional belief itself (Munro, 1982). The prognosis of patients with MHP who respond to pimozide is uncertain: while a partial or complete improvement can be expected in about 80% of correctly diagnosed cases, the majority will require indefinite maintenance treatment (Munro, 1982).

Although Ms U's clinical profile closely resembled that of the cases described in Munro's (1982) series, her response to treatment differed in a number of aspects. Whereas major depression is not uncommon in the post-psychotic phase of patients responding to pimozide, her recovery was followed on both occasions by brief episodes of mild hypomania. The daily dose of pimozide required to obtain symptom relief in MHP varies between 2 mg and 12 mg, and improvement usually occurs within a week or two. Ms U initially showed a similar response pattern but, following her subsequent relapse, required more than double her original dose of pimozide to regain complete remission. Her post-psychotic hypomania was probably due to the combined effect of intense relief after years of distress and the stimulating properties of pimozide, but to account for the higher dose of neuroleptic is more problematic. About a third of Munro's MHP patients reported a specific life event as a precipitating factor and he noted that those cases successfully weaned off medication remained vulnerable to relapse as a result of subsequent stress. In predisposed individuals a process analogous to 'kindling' may, therefore, occur in which repeated adverse experiences facilitate their susceptibility to future stress-related relapses (Munro, 1982). An alternative but perhaps complementary hypothesis is a 'supersensitivity psychosis', induced by chronic neuroleptic treatment (Chouinard & Jones, 1980). Patients exposed to a certain amount of daily stress may also be more likely to exhibit tolerance to the antipsychotic effects of neuroleptic drugs. On this basis one would predict that any future psychosexual difficulties could reawaken Ms U's symptoms, particularly if her medication was withdrawn altogether.

Phenomenologically, Ms U's belief had the characteristics of an overvalued idea, but her symptoms remained refractory to a variety of treatments other than pimozide. This supports the contention that the underlying psychopathology was that of a monodelusional pseudocyesis, resembling a hypochondriacal variant of Kraepelinian paranoia.

Acknowledgements

The author wishes to thank Professor Alistair Munro for his encouragement and advice and Professor Nils Retterstøl and Dr Stein Opjordsmoen for drawing his attention to the relevant Scandinavian literature. Professor Mogens Schou, Dr Krystyna Szulecka and Ms Helena Zymonová very kindly provided translations of the Danish, Polish and Czechoslovak literature.

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British Journal of Psychiatry (1990), **157**, 929–939

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Editor: Ian Pullen

Contents: Personality and outcome of depression/Violence in Alzheimer's disease/Oestrogen therapy and the menopause/The 'new cross-cultural psychiatry'/Ethnic minorities and the psychiatric system/Benzodiazepine withdrawal/Limitations of double-blind trials/The Lomax affair/Education for the 21st century/British Neuropsychiatry Association/The Bowdlerisation of psychiatry/Social order/mental disorder/Treatment of depression with pumpkin seeds/Down's syndrome, dementia and myoclonic jerks/How informed and binding is informed consent.

Personality and outcome of depression

SIR: Andrews *et al* (*Journal*, July 1990, **157**, 13–18) present a very interesting study of personality and long-term outcome in depression, but there are alternative explanations to the data presented.

The authors follow the tradition of regarding endogenous and neurotic depression as a dichotomy. Their study shows the negative influence of personality on the outcome of neurotic depression (ND) but not endogenous depression (ED). Andreasen has indicated factors which support a dichotomy view (Andreasen, 1980). The findings by Professor Andrews *et al* exemplify one of these.

The paper also suggests that endogenous and neurotic depression have different courses with 'episodic' or 'more insidious' features respectively. The data presented is open to different interpretation. A total of 57% of their ON group (other neurosis presenting as depression) have drifted into ND by the end of the study. This suggests a high degree of overlap between the two groups. It is therefore just as reasonable to sum the two groups together as to make a possibly artificial division between them. Doing this the combined ND+ON group may then be compared with the ED group. In the combined group ($n=127$), 16% remained well, 71% suffered a recurrence of some degree and 18% were incapacitated during the follow-up period. In the ED group ($n=66$) these figures are 15%, 60%, and 16% respectively. These findings seem more remarkable for their similarities than their differences. From a clinical perspective both ED and ND appear to have similar courses.

The authors also suggest that the characteristics of ND more closely resemble ON than ED. In view of the diagnostic drift from ON to ND this is perhaps not surprising.

In the view of the Newcastle group which Professor Andrews *et al* favour, a personality inadequacy is one factor defining neurotic rather than endogenous depression (Carney *et al*, 1965). This study demonstrates that in depression, where personality factors are initially an aetiological factor, they remain a factor influencing the future recurrence of illness. Where personality factors are less important, in an initial endogenous diagnosis, they remain less of an influence in the outcome of the illness.

Overall, the courses of ND and ED appear only a little different in outcome. What differences there are can be explained by the modifying effects of personality. The case for a dichotomy is not proven.

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CARNEY, M. W P, ROTH, M. & GARSIDE, R F (1965) The diagnosis of depressive syndromes and the prediction of ECT responses *British Journal of Psychiatry*, 3, 659–674

AUTHORS' REPLY: We have published two papers on the long-term outcome of our cohort of depressive patients (*Journal*, June 1988, **153**, 752–757 and *Journal*, July 1990, **157**, 13–18). Your correspondent appears to have overlooked the former paper in which we report that the morbidity at 15 years was the same for patients with endogenous or neurotic depression We thank your correspondent for restating what we have already published and indeed stated again in the abstract of the second paper: "diagnosis at index admission did not predict overall outcome" (1990, p.13). We noted that despite the similarity in outcome, "patients with endogenous depression, an apparently stable diagnosis, had longer index admissions, were readmitted sooner, but spent less time ill than patients in either of the neurosis groups" (1990, p.13). Personality abnormality as judged at index admission accounted for 20% of the variation in these measures in the neurotic but not in the endogenous group.

We have long argued that attempting to establish the independence of two syndromes on the basis of symptoms alone is foolish (Andrews *et al*, 1973). Syndromes that appear clinically distinct should be differentiated on the basis of aetiology, treatment response and/or natural history. In the present paper we simply report subtle differences in natural history, but not outcome, and relate these to a presumed aetiological factor (personality) that seems to act differently in the two disorders. Parker *et al* (1987) have previously shown that the parenting of our patients with endogenous depression differed from the parenting of patients with neurotic depression and that this was unlikely to be a reporting artefact.

We agree with Dr Aspin that "the case for a dichotomy is not proven", but there are now three pieces of evidence derived from just this one study that "endogenous and neurotic depression are two illnesses". We claimed no more.

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Violence in Alzheimer's disease

SIR: The series of articles on psychiatric phenomena in Alzheimer's disease by Burns *et al* (*Journal*, July 1990, **157**, 72–94) was illuminating. In this letter I would like to raise two issues.

First, the use of χ^2 test with some analysis appeared to be inappropriate as the expected frequency in the cells of some of the χ^2 tables was less than 5. Examples of this included Table II on p. 74, Table III on p. 84 and Table IV on p. 89. The more appropriate test would have been the Fisher's exact probability test (with a unidirectional hypothesis).

Second, the above study reported that 20% of patients exhibit aggressive behaviour and this figure is comparable with that of other studies (Swearer *et al*, 1988). Some of my own unpublished work had indicated comparatively low levels of violence (seven violent incidents in one year) on a long-stay psychogeriatric ward. The reported relationship between aggressive individuals and presence of temporal lobe atrophy on neuroradiology is fascinating. This relationship is consistent with reports of violence and aggression associated with electroencephalogram (EEG) abnormalities focused on temporal lobes (Tunke & Dermer, 1977). There are other reports of association between violence and aggressive behaviour and abnormal EEGs (Maletzky, 1973; Shah, 1989). The latter study was among mentally handicapped patients, a population which may be comparable with Alzheimer's disease patients.

There are reports of successful treatment with phenytoin (Maletzky, 1973) and carbamazepine (Tunke & Dermer, 1977) of violent and aggressive patients without overt epilepsy but with non-specific EEG abnormalities. There are also reports of successful treatment with carbamazepine of aggressive and violent behaviour among patients with dementia (Au Essa, 1986).

Aggressive and violent behaviour is not uncommon in Alzheimer's disease, causes considerable distress among carers, and is often an indication to admit to a long-stay psychogeriatric bed, with consequent iatrogenic disorders. In view of this one wonders if there is a case to use carbamazepine in such patients, which is increasingly used in psychiatry and is relatively free of side-effects (Tobiansky & Shah, 1989). At the very least there must be a strong case for an appropriately controlled trial of carbamazepine in such patients.

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AUTHORS' REPLY: We are grateful to Dr Shah for his remarks about our paper. With regard to statistical tests employed, the χ^2 test was the appropriate statistic as the sample size was greater than 20. A Yates correction to the χ^2 is the correct statistic when the expected frequency of at least one of the cells is less than 5. There is disagreement among statisticians as to whether such a correction is necessary (Everitt, 1977), and on statistical advice we did not apply such a correction. However, even after applying such a correction, the results remain significant for the association between left basal ganglia calcification and delusions, and just fail to reach significance ($P<0.06$) for the right side. We admitted in the discussion that the number of patients involved is small, but this makes the trend of the association between delusions and basal ganglia calcification even more interesting.

With regard to Dr Shah's second point, his own unpublished figures are not comparable with ours since they are based on a hospital sample whereas our study included patients in a variety of settings. We agree fully that adequate drug trials, possibly using medication such as carbamazepine, are important for the treatment of aggression – a very troublesome and disruptive behaviour in Alzheimer's disease.

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Oestrogen therapy for the menopause

SIR: Ballinger in her paper on psychiatric aspects of the menopause (*Journal*, June 1990, **156**, 773–787) comes to a clear conclusion that "anxiety and depression in such women do not respond to oestrogen therapy although some cases respond to antidepressants". Few endocrinologists and gynaecologists with a large experience in treating such women will agree with this dismissal of the value of oestrogens and we are sadly aware that there is virtually no meeting point between our specialties on this issue. This review article, quite devoid of objectivity and omitting most relevant publications during the last ten years, will reinforce traditional psychiatric prejudice on this subject.

"The gynaecological view" described by Dr Ballinger is a travesty which gives a biased view of what gynaecologists are meant to think. As one of us (JS) is singled out for particular scorn for "uncontrolled" statements, may we be permitted to bring to the attention of your readership some of the controlled studies of the value of oestrogens performed in this department and elsewhere that have been omitted by your author.

We would all agree that there is an excess of depression in women compared with men and that this excess begins from puberty and occurs more commonly at times of hormonal flux. This appears as pre-menstrual depression, post-natal depression and climacteric depression. Certainly these may also be times of greater stress and we accept much of the psychiatric emphasis on the environmental contribution to these peaks of psychiatric pathology but not to the exclusion of a hormonal component.

Dr Ballinger uses the term 'menopause' to describe the years around the time of cessation of periods. Although this is more precisely called the climacteric, it is acceptable in this context because of common usage and because, as she has stated, the literature suggests that any alleged increase in depression in these women occurs in the few years before the menopause. There is no clear evidence of an increased incidence of depression in women who are truly post-menopausal.

Depression in the 40-year-old woman with regular menstruation is frequently cyclical with attendant cyclical symptoms of loss of energy, loss of libido, bloated feelings, mastalgia and menstrual headaches. This combination of debilitating symptoms, best called the 'ovarian cycle syndrome', is common and gets worse with age until it becomes indistinguishable from the most distressing symptoms of the climacteric. The symptoms frequently lose their cyclicity, becoming more constant, but the similarity to those of the climacteric supports the view that the psychiatric symptoms of the latter are a continuum of those of the pre-menstrual syndrome. Oestrogens are effective in treating both conditions. The problem is that depression (although it may be cyclical) in the presence of regular periods is not considered by psychiatrists to be responsive to oestrogens.

Montgomery *et al* (1987) in a placebo-controlled study reported the beneficial effect of oestradiol implants on depression in a group of women attending our menopause clinic. However, this improvement in depression and anxiety was only seen in the pre-menopausal patients and not in those in whom periods had ceased. Data yet to be published show that this significant improvement in scores on the self-rating scale of distress (SRD-30) persists over two years, and Brincat *et al* (1984) have shown a recurrence of depression and other related symptoms when patients well controlled with implants for many years are then given a placebo. The same percutaneous implantation of 100 mg oestradiol every six months was used to treat depression in younger women with pre-menstrual syndrome and was found to be more effective than placebo in every Moos cluster of symptoms including negative affect (Magos *et al*, 1986). This is not transient as a sustained improvement has been reported after five years of such therapy (Watson *et al*, 1990).

Oestradiol implants have a prolonged duration of action which may be undesirable in some patients. Transcutaneous oestradiol patches in high doses (Estraderm, 200 µg) which do not have this long-term characteristic have also been studied with equally impressive effects on depression when compared with placebo (Watson *et al*, 1989).

These findings are not unique to our clinic but the space and number of references permitted in this letter do not allow me to give details of data from Montreal, Cardiff and London which support this view.

All doctors who treat depressed peri-menopausal women, regardless of the finer definitions of depression, should be aware of the potential that oestrogens have in relieving the suffering of some (or many) of these women. Nobody makes any claims that oestrogen therapy is a panacea for all the psychiatric problems of middle age, but patients deserve that the place of this therapy is evaluated carefully and not dismissed in such an unscholarly review.

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AUTHOR'S REPLY: Dr Studd and his colleagues express some dissatisfaction about the choice of references in my review, but I can only reiterate that the majority of general population studies do not support the view that the menopause or climacteric is associated with a significantly increased risk of psychiatric disturbance in women. In addition, the majority of treatment studies do not support the view that oestrogen has a specific antidepressant effect, but many women feel better if their symptoms of flushing and sweating are effectively relieved and this complicates the interpretation of many studies.

The 'ovarian cycle syndrome' described by Dr Studd and his colleagues and said to be common is difficult to evaluate in that the physical symptoms described may well make a woman feel depressed and, conversely, a depressed woman may be much less tolerant of, and more disturbed by, these cyclical changes in sensation. Symptoms such as loss of energy and loss of libido are certainly very common in straightforward depressive illness.

Several studies have shown that women attending gynaecology out-patient clinics have higher levels of psychiatric morbidity than matched controls from the general population (Munro, 1969; Worsley *et al*, 1977; Byrne, 1984). Dr Studd and his colleagues will see many depressed women who benefit from their clinic attendance. However, I would question the view that oestrogen implants are the main therapeutic agents as far as psychiatric symptoms are concerned and would suggest that it is the very obvious concern and enthusiasm of the staff that is of prime importance, as suggested in the study of Strickler *et al* (1977).

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The 'new cross-cultural psychiatry'

SIR: Littlewood (*Journal*, November 1990, **157**, 775–776) has misidentified a 'conventional error' in my editorial. I did not suggest that culture should be held constant. I was concerned with the extent to which folk concepts of mental illness need to be ascertained before embarking on any comparative study involving psychiatric symptoms and/or diagnosis. While it may be an ideal counsel to recommend this groundwork before any comparative psychiatric study is undertaken, it is simply not practical and will not be followed. I introduced my example to suggest that differences in folk concepts of mental illness between native whites of Salford and London are unlikely to be of a magnitude to invalidate the use of the same criteria for psychiatric symptoms and diagnosis across the populations. This strategy does not of course exclude the study of other cultural differences between the two populations.

On another issue, it is Dr Littlewood's perception that I have 'put down' local Yoruba knowledge about smallpox. This is a clear illustration of the different value systems held by anthropologists and medical practitioners (although Dr Littlewood is both, in this instance he is taking an anthropologist's stance). As discussed in my previous letter (*Journal*, August 1990, **157**, 296), an anthropologist is neutral as to whether or not people die of smallpox. His or her professional concern is with the local meaning of rituals and their value to the society that practices them. By contrast, the primary concern of the doctor is the prevention of disease and death, and he or she needs to determine whether a practice/ritual is effective in this respect. This by no means always involves 'putting down' non-Western practices. For over 2000 years Ayurvedic practitioners in India were using an effective antipsychotic agent, Rauwolfia, while doctors in the West were beating psychotic patients, chaining them and, even into the present century, immersing them in cold water. The meaning of these practices/rituals would be of interest to a social anthropologist, whereas the primary concern of a psychiatrist is that these ineffective methods of treatment have been replaced by effective ones, initially Rauwolfia.

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Ethnic minorities and the psychiatric system

SIR. Although we are somewhat uncertain how to acknowledge Littlewood's assessment of our paper (*Journal*, March 1990, **156**, 373–378) as "not unuseful", we are grateful to him for his less ambiguous critical comments (*Journal*, September 1990, **157**, 451–452). We agree that our study does not address the question of subtle or unconscious racism, but this was not our purpose. One of the starting points for our investigation of police admissions to psychiatric hospitals was to find out how much agreement there was between the policeman and psychiatrist in diagnosing mental illness in emergency referrals. Our results revealed a very high degree of concordance. One of the reasons that we undertook this investigation was in response to an assertion, supported by Dr Littlewood, that "the police are *overtly* racist and selectively pick out non-mentally ill black people in the streets and take them to a psychiatric hospital under Section 136 of the Mental Health Act as an alternative to arrest" (Littlewood, 1986). We believe that our study, carried out in an area with one of the highest Section 136 admission rates in the UK, challenges this assertion, and raises the concern that *overt* racism is more likely to result in mentally ill subjects from the ethnic minorities being channelled into the criminal justice system instead of the psychiatric services.

We appreciate the value of case vignette studies of the type advocated by Dr Littlewood and used in the study by Lewis *et al* (*Journal*, March 1990, **157**, 410–415). However, we are concerned that such an enlightened approach should not preclude descriptive studies which detail the progress of the ethnic minorities through the psychiatric services and the criminal justice system. It could even be argued that the results of such an approach (e.g. Harrison *et al*, 1988), which undoubtedly appear naive to the anthropologist, have brought transcultural psychiatry research to life in this country.

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Benzodiazepine withdrawal

SIR: We thank Hawley for his kind comments on our paper (*Journal*, November 1990, **157**, 777–778) and welcome the opportunity of replying.

Ideally, we should have liked to have included four treatment groups in our study: abrupt and gradual withdrawal, each with and without propranolol. Such a study was, however, impractical for us at the time. We therefore restricted it to the two groups described in our paper, as these seemed to be the most widely used and effective treatment regimes at the time the study was launched. We agree that our study does not dismiss the possibility of propranolol being of some use in benzodiazepine withdrawal in some patients. However, it is of note that in our sample, patients slowly withdrawn were successful in their efforts at withdrawal without suffering significant withdrawal symptoms. In the slow withdrawal group, therefore, few patients would have needed any adjunct to the slow withdrawal regime. It is also notable that in the abrupt withdrawal group, the addition of propranolol did not prevent patients from suffering considerable withdrawal symptoms that in many cases led to failure to complete the withdrawal process.

We intended to determine whether the advantages of slow withdrawal could be offset by using propranolol, thus allowing abrupt withdrawal to be successful in a similar proportion of cases. We believe that we have shown that this is not the case.

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Limitations of double-blind trials

SIR: Double (*Journal*, August 1990, **157**, 300) appears to have misunderstood the argument I put forward (*Journal*, February 1990, **156**, 282). The valuable article by Kramer & Shapiro (1984) he cites does not denigrate the value of the randomised controlled trial, but indeed upholds it as the method of choice for evaluation of therapeutic interventions, although pointing out several issues that are important to the design, execution and interpretation of a clinical trial. One of these issues is the possibility of bias arising when the desirable property of double-blindness is not attained, a possibility I certainly accept as impairing the interpretation of the results of a study.

Kramer & Shapiro suggested a strategy of asking participants to guess, on completion of the trial, which treatment they received, but solely as a means of assessing possible bias due to unblinding. They explicitly upheld the principle of analysis by 'intention to treat' and did not advocate stratification of the statistical analysis by blinding status. In a trial of ascorbic acid for the common cold, cited both by Dr Double and by Kramer & Shapiro, Karlowski *et al* (1975), having found that many more participants guessed their treatment correctly than guessed incorrectly, went on to examine evidence for efficacy in two subgroups separately, those who 'knew' which treatment they had received and those who 'did not know'. These subgroup labels were in fact misleading: the 'knew' group consisted of 79 subjects who guessed correctly; the 'did not know' group comprised not only 88 who did not offer a guess but also 23 who guessed incorrectly. This suggests that some 23 of the 79 who guessed correctly were doing no more than guessing; the 'did not know' group would more appropriately be supplemented by transferring to it 23 of the 'knew' group, either chosen randomly or by a weighted analysis.

Failure to demonstrate an advantage for active treatment in the 'did not know' group, as occurred in Karlowski *et al*'s study, need not imply that the benefit observed in an intention-to-treat analysis was illusory, for such a failure can arise in three ways. Firstly, the 'did not know' group may not be defined symmetrically (i.e. to contain as many correct guesses as incorrect ones). Karlowski *et al* failed in this respect, and the recommendations of Oxtoby *et al* (1989) seem to suggest doing just what Karlowski *et al* did. Secondly, problems can result from predominantly correct guessing, arising because of therapeutic efficacy. Karlowski *et al* were not able to exclude this and Oxtoby *et al* would be careful to distinguish it from guessing on the basis of side-effects. Thirdly, failure can arise from the depletion of the number of subjects included, and consequently of the statistical power to demonstrate a difference in effectiveness between treatments. This can seriously impair ones ability to demonstrate a null difference, as well as one's ability to demonstrate a substantial one.

The implication is clearly that the issue of maintenance of blindness should be dealt with as far as possible by good study design and execution. This will not always avoid the problem, but when it does, it will be much more satisfactory than a somewhat artificial salvaging operation in the statistical analysis. Often the results of a trial will fail to be as definitive as one would like, but will generally be subject to fewer limitations than inferences from purely observational studies. Interpretation of results is inevitably of the greatest importance; equally inevitably, the scope for controversy of interpretation will always be particularly wide in psychiatry.

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The Lomax affair

SIR: Budden (*Journal*, August 1990, **157**, 301–302) is concerned that my account of the Lomax affair (*Journal*, February 1990, **156**, 180–187) gives the impression that asylums were "places of brutality and inhumanity in general". He believes that the unusual conditions prevailing at the end of the first world war were responsible among other things for the high death rate among patients. Lomax was well aware that the shortages because of the war created great difficulties for asylum management. However, his indictment of asylum administration went much further than accounts of brutality, malnutrition and a high mortality rate. He believed that the English asylum system had become a closed-off world, with ineffective outside control, which existed "merely to confine the insane". Senior and presumably well informed civil servants in the Ministry of Health did not believe that the war was a sufficient explanation for the appalling conditions: Mr (later Sir Percy) Barter wrote, in a confidential minute to the Health Minister on Lomax's criticism, "... allowing for irregularities due to war conditions, the indictment is I believe in the main well founded".

My reason for unearthing the Lomax affair was not to open a debate about how good or bad conditions in English asylums were over 70 years ago. Rather, I was interested in how changes in mental health services are brought about and the role of an outsider who chose to challenge the psychiatric establishment. By 1924, all observers agreed that major reforms were necessary.

The editorialist in the *Lancet* commenting on my paper justifies reviving the Lomax affair thus: "... injustice is always wrong, and it is better to put it right seventy years later than to let it persist". She or he comments further that in the face of the current neglect and ill treatment of mentally ill people in the community, "Britain may need another Montagu Lomax in the 1990s, with a wider remit" (Editorial, 1990).

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Education for the 21st century

SIR: Cawley (*Journal*, August 1990, **157**, 174–181) is correct in drawing our attention to the education of the psychiatrist of the 21st century. His remarks on the role of the Collegiate Trainees Committee (CTC) are thought-provoking. Indeed, in the past 11 years of its existence the CTC has put forward many new ideas of which some have been accepted by the College rather quickly, and others abandoned quietly. The acceptance or the rejection has not depended on the goodness of the idea but the 'goodness of fit'. This goodness of fit is what we would call 'fitting in with the system'. However, this process of 'fitness' depends upon not only the activity of the trainees but also the receptivity of the College. Unfortunately, the CTC does not have enough power in its own right to put through changes. Also, the inexperience of trainees in the political arena means that even though they may put forward fresh thinking and ideas in an enthusiastic manner, their naivety and lack of political clout and often lack of support among senior colleagues may cost them dearly. From local experience, it is apparent that trainees often are scared to put their names on paper in favour of anything that may be perceived controversial and thence prejudicial to their careers.

Certainly over the last few years, the CTC has led the way on many issues. Public image of psychiatry was discussed even before the Public Education Officer was appointed by the College. The role of training in community care settings was put forward by the CTC. Of the seven points that Professor Cawley has raised, the CTC has specifically looked at the practice of psychiatry in primary care, the role of management and audit in training, part-time training for doctors with domestic commitments and the role of ethics in psychiatry. The CTC led the way with its working party reports on the training of psychiatrists in Europe, and the College is now involved in setting up a European conference scheduled for 1992.

To the best of our knowledge this is the only committee in the College that takes women's issues seriously and has the subject as a standing item on the agenda. We are moving forward slowly but surely, and we ask of our colleagues – senior and junior – to support us when we are right and to correct us if we are not. The CTC cannot be a constant pain simply because the habituation response would decrease its value. An occasional irregular jolt is more likely to keep the College on its toes.

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British Neuropsychiatry Association

SIR: Dening (*Journal*, June 1990, **156**, 915), in his review of essential reading in the field of 'organic psychiatry', is kind enough to suggest that psychiatric trainees should consider joining the British Neuropsychiatry Association. We, the committee of that Association, are indeed keen to encourage all interested trainees to join. The British Neuropsychiatry Association (BNPA) was formed in 1988 in order to encourage cross-disciplinary discussion in the fields of neuropsychiatry, neurology, neuropsychology and allied basic sciences. So far, we have held meetings twice each year, each of which have concentrated on the neuropsychiatry of some particular topic, such as the frontal lobes, movement disorders, dementia and emotion. Speakers have always come from a wide range of neuropsychiatric and allied disciplines, and we encourage interesting case and video presentations, particularly by trainees. It has generally been agreed that our meetings are lively, varied and interesting.

Membership of the Association costs £15.00 per annum and attendance at our meetings is free to members. If trainees (or any other psychiatrists) are interested, then we hope that they will feel free to contact any member of the committee for further information. In particular they should contact the Honorary Secretary, Dr J. M. Bird, Consultant Neuropsychiatrist, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol BS16 1QT. (Tel: (0272) 701212 ext. 2925).

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The Bowdlerisation of psychiatry

SIR: Lewis and his colleagues have recently published two articles in the Journal based on postal surveys of psychiatrists' responses to case vignettes. These reports show disturbing stereotyping by psychiatrists.

In the first article (Lewis & Appleby; *Journal*, July 1988, **153**, 44–49), perjorative judgements turning on the previous diagnosis of a personality disorder were used to argue that the concept of personality disorder "should be abandoned". In the recent article (Lewis *et al; Journal*, September 1990, **157**, 410–415) entitled "Are British psychiatrists racist?", essentially similar results, turning on the words 'white' versus 'Afro-Caribbean', lead them to suggest that the concept of racism be avoided and that of 'race thinking' be substituted. Abandoning the concept of 'black' would be more consistent but more problematic.

In the latest article (paragraphs 2 & 3, p. 415) Dr Lewis *et al* suggest that the "professional literature and training", in particular their own contribution to the former, may prevent prejudice "by highlighting the nature of this stereotype". We wonder about their understanding of the word 'highlighting'.

Tomas Bowdler, MD (1754–1825), published his *Family Shakespeare* in 1818. It was an "expurgated edition". He went on, perhaps less influentially, to remove all sexual references from Gibbon's *Decline and Fall of the Roman Empire*. Lewis and his colleagues suggest a similar semantic purification of the psychiatric literature, but their Bowdlerisations are neither consistent nor a profitable solution. To remove or tone down words is not to 'highlight' stereotypes and their dangers. Perhaps caring professionals find it as painful to acknowledge their prejudices and unconscious racism as the Victorians found it to acknowledge sexual intercourse.

Dr Lewis *et al* suggest that accusations of racism "seldom change beliefs or behaviour for the better", but neither will abandoning terms nor the use of comfortable phrases like 'race thinking'. These strategies do not expunge the obnoxious attitudes. A sample of British psychiatrists would more often expect an Afro-Caribbean patient to be violent than a white patient and were more likely to advocate criminal proceedings against the Afro-Caribbean patient. Lewis *et al* pose the question "Are British psychiatrists racist?" but never give the answer, clear in their own findings, "yes".

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Social order/mental disorder

SIR: While I was pleased to see a review (*Journal*, March 1990, **156**, 454–455) of my most recent book, *Social Order/Mental Disorder* (1989), I found much in the review itself with which I would wish to quarrel. I shall not dwell on matters of taste and interpretation, since these are, as Henry Rollin acknowledges, matters on which dispute can be endless. Besides, criticisms of this sort are perfectly legitimate matters for a reviewer to raise. But I do object to having my position misrepresented and caricatured in order to provide the reviewer with a straw man to assault.

I must first take issue with the peculiar notion that it is the historian's duty to preserve the reputation of the "folk heroes of British psychiatry" intact, heedless of what the historical record may show; and without belabouring the point, I think that most people outside the ranks of organised psychiatry would raise an eyebrow or two at the notion that psychiatrists themselves (and psychiatrists alone?) qualify as "historians whose impartiality is unimpeachable". But, more particularly, I must protest at the absurd claim that I argue that the proliferation of 19th century asylums "was engineered by unscrupulous doctors" – and at the still stranger notion that I am "a staunch advocate of community care" and view the late 18th-century as "the Golden Age for the mentally disordered".

Lunacy reform in the Victorian age was a long and complicated process, in which medical men played a significant but clearly subordinate role. The great importance of other historical factors and actors (including the "redoubtable Earl of Shaftesbury") is scarcely news to me, since I have published a lengthy monograph on the subject (Skull, 1979). No careful or "impartial" reader of either that extended analysis, or of the necessarily more fragmentary discussions of the subject in *Social Order/Mental Disorder*, would, I submit, recognise Dr Rollin's caricature as a fair representation of my position.

As for "community care", there is something more than slightly ironic about the claim that I am one of its cheerleaders. My *Decarceration* (Skull, 1977) was one of the first sustained critiques of deinstitutionalisation. In *Social Order/Mental Disorder*, I return repeatedly to "the appalling deficiencies of yet another generation of 'mental health reforms' "; I refer to the "castastrophic failures masquerading under the official guise of a 'revolution' in psychiatric care"; criticise "the odd mixture of zealots and penny-pinching politicians who continue to call malign neglect 'community care' "; document that "community care is simply a sham"; and analyse why such an approach nonetheless continues to dominate mental health policies on both sides of the Atlantic. So far from romanticising the 18th century, I specifically compare the contemporary board and care industry in the United States, which has battened upon the legions of discharged mental patients, with the horrors of the late 18th-century English madhouses or "trade in lunacy". I call attention to "our collective reluctance to make a serious and sustained effort to provide a humane and caring environment for those manifesting grave and persistent mental disturbance"; and, dare I say it, I protest "the retreat even of organised psychiatry from any attempt to deal with their problems".

Dr Rollin's bee in his bonnet about sociologists who work on the history of psychiatry has evidently precluded him from reading what I actually have to say We have plenty of real issues to debate, for I am far from being a complacent or uncritical observer of the psychiatric scene, past or present. But it would help if we could argue about matters of substance, rather than engaging in disciplinary demonology.

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Treatment of depression with pumpkin seeds

SIR: I suspect that many colleagues may have shared my experience of patients who were previously maintained on L-tryptophan but have suffered partial or complete relapse since its withdrawal from the market earlier this year. I report on one such case and his successful treatment with pumpkin seeds.

Case report: A 44-year-old man of Polish descent first presented to the psychiatric services in 1982 with recurrent unipolar depressions. His subsequent treatment was complicated by his suffering significant side-effects on most first-line antidepressants. Those that he was able to tolerate (e.g. low-dose clomipramine) tended to induce hypomania after a few weeks of treatment. It was in some desperation, therefore, that I put him on L-tryptophan in 1985. Such was his apparent sensitivity to psychotropic medication that he became hypomanic on 4.5 g of L-tryptophan per day. During the next few months he became very stable on 1·5 g of L-tryptophan per day and he was discharged from follow-up in July 1985.

He was referred back by his general practitioner this August. It seems that he had remained entirely well over the five years previous to this, apart from a bout of depression with biological features every three months or so. At these points he took about 1·5 g of L-tryptophan over two days and found that this quickly restored his well being. When he was referred back to me he had been constantly depressed for three months with anergia, early-morning wakening, loss of appetite and anhedonia.

Another patient (a medical practitioner) who had suffered sleep disturbance on withdrawing from the L-tryptophan on which she had been maintained, had researched the topic and established that pumpkin seeds were the most cost-effective means of administering natural L-tryptophan: a typical 250 g bag contains 1300 mg and costs about £1.00.

Within 24 hours of ingesting about 200 grams of pumpkin seeds (i.e. about 1 gram of L-tryptophan), the patient felt quite transformed. He was no longer anergic or depressed and happily returned to work the following day.

Clearly this patient is atypical in that his mood state appears to be remarkably sensitive to L-tryptophan. However, for patients who were successfully maintained on L-tryptophan or for whom one wishes to instigate L-tryptophan, pumpkin seeds may represent the best alternative at present. Perhaps, as in this case, the biggest hurdle is overcoming the scepticism of one's patient!

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Down's syndrome, dementia and myoclonic jerks

SIR: The recent publicity surrounding bovine spongiform encephalopathy (BSE) has intensified the search for cases of the human concomitant, Creuzfeldt Jakob disease. The combination of a dementing process and myoclonic jerks in a person under the age of 70 years raises suspicions of such a diagnosis. I urge for caution before reaching this conclusion.

There are two accounts in the literature of a combination of Down's syndrome, dementia and myoclonus (Blumbergs *et al*, 1981; Good & Howard, 1982). Both were originally presumed to be Creuzfeldt Jakob disease but post-mortem findings indicated Alzheimer's disease which commonly occurs prematurely in people with Down's syndrome. Faden & Townsend (1976) draw attention to the confusion arising when myoclonus occurs in association with dementia, and assert that this should not be considered pathognomonic of Creuzfeldt Jakob disease and that a diagnosis of Alzheimer's disease should be considered. This difficult diagnostic problem is illustrated by the following case.

Case report: A is a 48-year-old single lady with Down's syndrome. She developed *grand mal* epilepsy three years ago and an electroencephalogram (EEG) at that time showed no evidence of a space-occupying lesion She was successfully treated with phenytoin and seizures are now infrequent.

For the last 18 months there has been a steady deterioration in her memory and she is now unable to recognise close family members. Her speech has deteriorated and she spends much of her time muttering to herself. Her self-care has deteriorated and she has become doubly incontinent, necessitating full nursing care. Her motivation is poor and her mood is labile. One year ago she developed myoclonic jerks. These occur only 1–2 hours after waking and affect her head, arms, shoulders, body and legs. They are abrupt, brief, irregular, asymmetrical and can be so severe as to catapult her out of her wheelchair.

A repeat EEG shows generalised irregular 3–7 Hz slow activity, with no change from her EEG of 1987. In particular, there is no evidence of periodic biphasic or triphasic complexes characteristic of Creuzfeldt Jakob disease

In the light of the experience of other authors, the time course of the clinical findings and the lack of specific abnormalities on the EEG of this patient, the diagnosis should be Alzheimer's disease until proven otherwise.

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How informed and binding is informed consent?

SIR: We report a case in which the patient's consent for an interview with him to be broadcast, given while he was well, had an affect on his mental state.

Case report: AB, a 37-year-old male homosexual teacher was diagnosed as having AIDS following pneumocystis carinii pneumonia. Initially, he accepted the diagnosis well but following a move to his new flat he became depressed. He withdrew from his friends and family, wished he were dead, but did not make any suicidal gestures. He was admitted to an acute psychiatric ward with the diagnosis of depressive illness, where he revealed that he was preoccupied with the idea that the interview he had recorded for a gay programme on television would reveal his sexual orientation and illness to the families of children he had taught previously. He feared that these families would harm him and the popular press would scapegoat him. He regretted having made the interview and had approached the television production team involved repeatedly, asking for his material to be withdrawn He was informed that they had gained his written consent for the interview to be broadcast and intended to proceed with it. Following admission and noting his mental state, the medical team contacted the programme director and requested that material involving the patient be edited. The director consented after receiving the letter. This certainly helped alleviate the patient's worries, and following antidepressant medication and psychological treatment the patient was discharged.

As doctors, we are often critical of the way in which the broadcast and newspaper media collect and report information about patients. We present an example of the media behaving in the responsible manner, in cooperation with the medical staff, and the beneficial effect of such an intervention upon the patient's view of life and mood. While we are heartened by one experience in this case, the issue of informed consent for the broadcast of material containing medical as well as psychiatric patients, seems to be an area in which the best interests of the patients are often compromised by the desire of the journalists to collect visual images to accompany their stories about ward closures, living in the community and advances in medical technology. There seems to be an urgent need to review the issues involved in protecting patients from the media (especially the ones who may change their minds and withdraw their consent) in a society where a free press often comes close to compromising a patient's right to anonymity and confidentiality.

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CORRIGENDUM

Journal, October 1990, **157**, 626. The full reference should read *Journal of Mental Science*, 1890, **36**, 150–151. "Morton Hospital" should read "Horton Hospital".

A HUNDRED YEARS AGO

"Traumatic neuroses"

This is a convenient term, but, like other terms which are convenient, it is apt to include far too much. In this country the most important traumatic neurosis which is met with is no doubt the so-called "railway spine", and it may be that cases placed in this category are as varied as those which Hoffman (Berlin. Klin. Woch., No. 29) found among a series of twenty-four cases of "traumatic neurosis". Of those twenty-four, ten were found to have undoubted signs of organic mischief; in six the symptoms were partly the result of exaggeration and partly of simulation; in eight there was malingering, proved to be so after careful observation for several weeks. The author protests against the use of the term for such varied conditions, pointing out that in a so-called traumatic neurosis we may have to deal with organic nerve injury, with hysteria, the result of injury, with shock to the cerebrospinal system, with neurasthenia, or even with a true psychosis.

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The Lancet, 17 January 1891

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

British Journal of Psychiatry (1990), 157, 940–943

Reading About . . .

Psychoanalysis

The reading I suggest first is concerned with the Oedipus complex, a major explanatory theory of psychoanalysis based upon a set of observations and crucial to the understanding of the personality and its relationships. It refers to an organised configuration of loving and hostile feelings which the child experiences towards its parents. It is the desire for the total possession of mother and the death of father as a rival, but is also love of the father and rivalry with the mother. Siblings, babies and genital and pregenital versions of sexual intercourse and masturbation enter into it as well. My brief description draws upon the discussion in Laplanche & Pontalis' (1973) invaluable dictionary of psychoanalytic terms and concepts.

For many reasons it is difficult to fully appreciate the power and scope of the Oedipus-complex theory. For instance, in the first half of life individuals can have the unnoticed belief that their oedipal phantasies can be realised. It is only with realisations of a different sort – of limitations, the inevitable loss of parents and the fact of personal mortality – that oedipal elements force themselves upon the personality in a way which demands the re-working of adjustments previously achieved in adolescence and childhood; more explicit recognition and insight requires a personal analysis.

Freud first put forward the theory of the Oedipus complex in 1897 during one of the most productive phases of his life. He was suffering at the same time from his own psychoneurosis with mood changes and states of intense anxiety. At the end of October 1896 his father had died. The period is described in the two main biographies of Freud, the classic by Jones (1953) and the more recent work by Gay (1988). Ellenberger (1970), in *The Discovery of the Unconscious,* mentions this period as part of what he terms Freud's creative neurosis. Ellenberger's work is a means of orientation in a difficult terrain. He describes the history of pre-Freudian notions of the dynamic unconscious, and of psychodynamic therapies, and shows where Freud's theories are situated in the history of ideas. As a general introduction to Freud's own ideas it is hard to better Jones' (1953) biography and Wollheim's (1971) concise account of Freud in the Fontana Modern Masters series. All Freud's biographers draw heavily upon the correspondence between Freud (1887–1902) and Wilhelm Fliess which reveals Freud's life, state of mind and way of working in the 1890s when some of his most important theories and methods of investigation were born.

One of Freud's means of discovery in this period was his self-analysis, a method which itself was a major achievement: many have entered into periods of intense self-examination and reorganisation with major results, but Freud managed to systematise something of the means of this process. His self-analytic procedure was modelled upon his therapeutic method with his patients but it, in its turn, contributed to the development of Freud's technique – psychoanalysis. Furthermore, the self-analysis was intimately linked both with his writing of *The Interpretation of Dreams* (1900), and with his bereavement ("... my reaction to my father's death – that is to say the most poignant loss of a man's life"), out of which the discovery of the Oedipus-complex theory came.

Freud's self-analysis was only sometimes a detached and intellectual self-scrutiny. Often, it was a powerful process such as develops in good analyses. This process is not contrived or willed but arises from powerful, non-voluntary sources deep within, and from this derives the conviction which attends these discoveries. Freud wrote of the turbulence and non-deniability of the analytic processes in a letter to Fliess (27 October, 1897): "... I am living only for "inner" work. It gets hold of me and hauls me through the past in rapid association of ideas; and my mood changes like the landscape seen by a traveller from a train". He goes on to quote Goethe: "And the shades of loved ones appear, and with them like an old, half-forgotten myth, first love and friendship". To this, Freud adds, somewhat ruefully, "as well as first terror and strife" (letter 72).

The insights gained in his self-analysis provided Freud with an understanding of the nature of family relationships and their internal representation, which enabled him to build a theory based upon motives and meaning. Freud was making a psychological theory in which the physiological principles that marked his earliest theories remained only as parallel and increasingly metaphorical structures.

The major conclusion of this period arose from Freud's growing disbelief of the memories of florid seduction by their parents revealed by many of his patients. He reasoned that, while the seduction of children occurred to a much greater extent than had been previously acknowledged, the memories of many of his patients were based on their wishful fantasies rather than on fact. In a letter to Fliess (15 October, 1897) he announced one factor which changed his view: "Only one idea of general value has occurred to me. I have found love of the mother and jealously of the father in my own case too, and now believe it to be a general phenomenon of early childhood even if it does not always occur so early as in children who have been made hysterics. The tragedy of Oedipus Rex endures... because it seizes on a compulsion which everyone recognises because he has felt traces of it in himself. Every member of the audience was once a budding Oedipus in phantasy, and this dream fulfilment played out in reality causes everyone to recoil in horror, with the full measure of repression which separates his infantile from his present state".

After interpreting one of the classical world's great tragedies (Sophocles, 429–420 B.C.) he moved on to consider Shakespeare's most difficult tragedy. Shakespeare depicts Hamlet, a very modern hero, in a state of confused and guilty bereavement. "Shades of loved ones" have appeared to Hamlet too. How can Hamlet's hesitation to avenge his father by killing his uncle be explained? Freud's answer: "How better than by the torment roused in him by the obscure memory that he himself had meditated the same deed against his father because of passion for his mother, "use every man after his desert, and who shall 'scape a whipping?' ".

Psychoanalytic insights can enrich our appreciation of the work of creative artists. The reverse holds with equal force. These great works, which can support so many different readings, enable us to contemplate the range of human passions which are also the raw data of psychoanalytic theories. Hamlet can help us understand what the theory of the Oedipus complex is really about.

The loss of loved ones leads to major upheavals. There is a re-working of deep and often ambivalent feelings. Every loss revives earlier losses and the powerful feelings of infancy are stirred; the Oedipus complex is the source of some of our very earliest loss. We all have to give up the exclusive possessive love for, and of, our parents. According to psychoanalysis, loss, as well as first love, is built in.

Freud's first observations about the Oedipus complex were more subtle than popular formulaic versions allow. It is well known that infantile sexuality, in the sense of infants and young children having diverse sexual excitements and phantasies, is considered to be important in personality development. However, Freud also concluded that children's observations of their parent's intercourse – the primal scene – can have a telling influence. And where there was no opportunity for observation, the child's imagination, his phantasy, would provide.

Freud further developed his ideas with observations about the internalisation of parental objects when oedipal wishes are repressed, and he considered this to be the essence of superego formation. In a very important paper, "Mourning and melancholia" (1917), he described how one part of the ego sets itself up in judgement over another, how melancholia and mourning are both linked with object loss and how the self-accusations of the melancholic are also complaints directed at an introjected object.

Klein's (1945) paper "The Oedipus complex in the light of early anxieties" gives an account of her work on the role of Oedipus situations in the development of boys and girls. Differing in many areas from Freud, Klein felt that oedipal fantasies occurred almost from the beginning of life; that the little girl had an awareness of herself as female, possessing an internal space and the vagina as a means of entry to it; that both boys and girls had an intensely possessive loving and hating relationship to the mother, first to her breasts but also, from very early, to her body and its contents, including babies; that the father, like the mother, is first experienced as a part-object; and that all these objects are felt to exist inside the child as well as outside. The feelings are at first extremely intense and the infant and very young child has periods when it veers from experiences of ideal satisfaction to those of intense persecution and distress.

Klein's description of her work with two children reveals the originality of her approach. There are sustained insights into internalisation and phantasy, two areas which she investigated in great detail. The notion of unconscious phantasy arose out of her technique with young children and was dependent upon her extraordinary capacity to appreciate the symbolic significance of play activities. Klein seeks to show how the perception and appreciation of the world and people in it is radically influenced by internalised object relations, and by the child's continuously active unconscious phantasy relationships, which also affect the state of the objects that the child unconsciously feels to be inside them.

The Oedipus Complex Today (Britton, Feldman & O'Shaughnessy, 1989), a statement of modern

Kleinian thinking, takes Klein's 1945 paper as its starting point. The three authors approach the Oedipus complex with different perspectives upon a shared point of view. The manifestations of oedipal situations can be subtle and difficult to spot, and these authors address instances where such qualities obtain. However, the clinical descriptions are also valuable because they show oedipal situations as ubiquitous, reassuringly commonplace and everyday.

Very sick patients are unable to negotiate oedipal situations in their development as this is dependent upon the internalisation of a secure good object as a central core to the ego. This process has miscarried in these patients, sometimes for environmental reasons, sometimes because of constitutional factors and always as a result of an interaction of both. O'Shaughnessy describes these patients in whom oedipal feelings are not evident because they are too intense to be faced. Of one occasion in the treatment of an 11-year-old boy she writes: "I said he felt I had mentioned his brother at the wrong time just when he was longing to have me to himself, and rage and disappointment were now pushing and pulling him about. I linked these feelings to his infancy, and how the baby that was still there in him felt that his mother, by becoming pregnant when he was four months old, filled herself with his brother at the wrong time, because he still needed to have her for himself."

Britton comments upon the shift of emphasis from the child's possessive sexual feelings to the impact of the child's awareness of the parents' sexual relationship. Some children are able to face this "challenge to the goodness of mother", and, after a period of mourning the loss of the previous exclusive, possessive relationship, re-establish a relationship with both parents on a new basis. Others cannot accomplish this, introducing some split in the ego, disintegrating or attacking the perceptual apparatus – their own minds — which registers the nature of the world.

Interestingly, the thinking process seems to be greatly affected by, for example, phantasies of controlling or taking the place of the parental couple. Feldman shows this through careful clinical descriptions. These emotional influences upon the thinking processes and upon cognitive development are more fully understood as a result of Wilfred Bion's work. Roger Money-Kyrle (1968, 1971) describes this work and the consequent far-reaching reformulation of psychoanalytic subject matter. Essentially, this can be conceived as the response of the infantile mind to certain basic facts and experiences. These include notions of the creativity of parental intercourse and of the capacity of the breast to nourish and nurture, which do not seem to be entirely dependent upon experience. Basic experiences and phantasies around issues of this sort provoke intense hostility and envy as well as love and admiration, resulting in the unconscious misrepresentations, delusions and misconceptions which are to some extent part of normal development. A corollary of Money-Kyrle's approach is that he can describe certain types of superegos as misconceptions.

Defining more precisely what psychoanalytic knowledge actually is has fascinated analysts, psychiatrists, critics and philosophers. Psychiatrists interested in analysis are often preoccupied with clarifying for themselves the epistemological issues raised by psychoanalytic ideas. What is the distinguishing quality of psychoanalytic findings? In my view it is an attempt to be objective about subjectivity while still describing what things look like and feel like from the inside.

In an essay, "Philosophy, metaphysics and psychoanalysis" the philosopher John Wisdom (1953) examines the distinction between discovery by reflection and discovery by investigation, and between knowledge about people with consciousness and knowledge about inanimate entities. He compares Freud's discovery which linked the unconscious and the repitition of the past with Newton's discovery of gravity which also connected already known phenomena.

Wisdom illustrates one type of psychoanalytic discovery with this example: "It's like someone who is trying on a hat and is studying it in a mirror. There is a pause and a friend says, "My dear, the Taj Mahal". All along she has known there was something wrong with the hat, now she sees what it is."

One reason why psychoanalytic findings are valuable is that meaning, mentality and emotionality are addressed in their own terms. This yields data about intangible entities. Anxiety has no shape or smell, yet we are in no doubt when we are in its presence. However, sometimes, it is worrying that in the last analysis there is still nothing to put a finger upon.

The direct observation of behaviour and interaction can provide more objective data which occasionally assuages some of these anxieties. If they are relied upon too much the distinctive subjective quality of analytic findings is lost. The observation of infants and children has a particular relevance for psychoanalysis with its reconstructive hypotheses about childhood and the idea that infantile factors continue to be relevant in the adult personality. One infant observation of great interest is that which Charles Darwin (1877) published in *Mind* of one of his own children.

Many analysts have devoted considerable efforts to the formal observation of development. Of course, while their studies were of theoretical value and empirical reassurance, they also had humanitarian effects. Rene Spitz, who in 1910 had a training analysis with Freud, in 1945 published a classic paper, "Hospitalism: an inquiry into the genesis of psychiatric conditions in early childhood", demonstrating the effects upon development of the existing pattern of care in institutions, particularly the lack of mothering and of a mother. Fraiberg, in a commentary upon Spitz's (1945) work, wrote: "The younger generation no longer knows first hand that when Spitz's studies of hospitalism and anaclitic depression appeared in the 1940s they were greeted in psychological circles with disbelief that deprivation of mothering could produce enduring effects on an infant's psychological development."

As a result of this work as well as that of Robertson and Bowlby in this country, attitudes to children, admission to hospital and adoption procedures have changed enormously. Moreover, these observations and the analytic work which preceded them have led to a harvest in the field of developmental psychology. Patterns of emotional reciprocity between mother and infant have been recognised as an essential part of the developmental process. Brazelton *et al* (1975) describe the mutual cycling of attention and affectivity which can be observed between young infants (2–20 weeks) and their mothers. They suggest that the goal of this is to achieve affective synchrony, and among its many powerful effects may be the modulation of excitement, mood and the taking in of the maternal environment. When mothers were asked to keep a frozen face for a few minutes their infants were very upset, looking hopeless and depressed. In infant–mother pairs who failed to thrive, Brazelton *et al* (1975) observed a "chaotic non-synchrony" and suggest that this embodied a basic distortion of "complex, expected and necessary rules for interaction". There are psychoanalytic contributions on the nature of mother–infant interaction. Winnicott's "holding" and Bion's "containing" are two important theories about emotional and mental operations and attitudes. In this area there is an opportunity for the integration of domains.

Psychoanalysis is a large subject with many avenues of approach and the reading recommended here is highly selective. Before leaving you to these highways and byways I recommend a paper by Riviere (1936), "A contribution to the analysis of the negative therapeutic reaction", which deals with those patients who are difficult to treat by any means, who respond to progress with deterioration, and who are narcissistically inaccessible. Riviere cautions against analysts using the negative therapeutic reaction in a self-exonerating way and shows why these patients are so threatened by change. Her description of the depressive anxieties to be faced in periods of psychic change is unsurpassed.

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Additional reading on psychotherapy

The Royal College of Psychiatrist's reading list on psychotherapy may also be of interest to readers.

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British Journal of Psychiatry (1990), **157**, 944–953

Book Reviews

Editor: Sidney Crown

Psychiatry for Students (7th edn). By David Stafford-Clark and Paul Bridges with Dora Black. London: Unwin Hyman. 1990. 320 pp. £11.95.

This textbook has now been available for over a quarter of a century. This seventh edition has managed to maintain the attractive, direct and clear style of the previous editions. The authors and editors are to be congratulated on bringing the text up to date, in keeping with the rapid changes that are occurring in the speciality. They have also managed to prune judiciously, thus keeping to manageable proportions what is primarily an undergraduate textbook. The references and bibliography are up to date, and the authors have concentrated on quality rather than quantity, which will greatly increase the possibility of students referring to the relevant research.

This is primarily a clinical textbook with excellent case vignettes which help to make the case histories come alive. This technique is one of the most effective ways of teaching a complex subject such as psychiatry. The textbook is divided into five sections: an introduction, the neuroses, the psychoses, miscellaneous disorders and management. The introduction includes an excellent chapter on history taking and the mental state examination, which students should find of great assistance during their clinical clerkship. The section on neuroses has a well written and informative chapter on personality development but is somewhat disappointing with regard to the contribution on anxiety and phobic states. I was disappointed that there was no reference to 'panic disorder' or post-traumatic stress disorder, although the latter was referred to in an excellent chapter on child and adolescent psychiatry.

In the section on psychoses I would recommend the chapter on organic disorders. The chapter on schizophrenia has been updated and refers to the new classification of schizophrenia (type I and type II). The epidemiology of schizophrenia is very well covered but its biochemical theories could have been described in more detail. In keeping with the increase in the elderly population and the current interest in psychogeriatrics, there is a welcome new chapter on disorders of old age.

Section four, which deals with miscellaneous disorders, has an informative chapter on mental handicap and a very well written chapter on child and adolescent psychiatry which will act as a sound clinical base for students as their first contact with this subspeciality. The chapter on alcoholism, although taking into account new developments in this field, is somewhat sparse in its coverage of this subject.

Section five has fully comprehensive chapters on psychological and organic treatments, psychiatric emergencies and psychiatry and the law.

In summary, this is an invaluable textbook which will serve medical students, nurses and interns as a good introduction to clinical psychiatry. The authors have updated the text without compromising the style of the original work. I recommend it to all students starting psychiatry and feel it is excellent value at £11.95.

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Medical Psychiatry: Theory and Practice – Volume 2. Edited by Enrique S. Garza-Treviño. Singapore: World Scientific Publishing. 1989. 668 pp. £39.00.

This is the second of two volumes, each with 13 chapters. The chapters of both volumes are listed in the contents, although no forward or preface is included. The book's presentation is disappointing since it consists of a compilation of camera-ready typescripts. Tables are at the end of each chapter rather than in the main body of the text.

It is difficult to know at whom the book is aimed. While the majority of chapters are concerned with some aspect of psychosomatic medicine or consultation liaison psychiatry, the inclusion of chapters on dementia and affective disorders in the first volume and benzodiazepines, encopresis/enuresis and clinical drug trials in the second is puzzling.

The first chapter is a brief overview of psychoneuroendocrinology with little relevance to subsequent, more practically orientated chapters. The next chapter on infertility is a useful update on a specific topic whereas the following two related chapters on sexual dysfunction and psychiatric aspects of obstetrics and gynaecology are simply reviews of very broad subjects.

Three chapters on malignant disease follow, illustrating the same inconsistencies. The first is a general review of psychological distress in cancer patients. The second is of an integrated model for psychotherapy developed by the authors, and the third deals with the neuropsychiatric complications of cancer and its treatments. An interesting chapter on organ transplantation follows in which the authors combine a general review with practical guidelines. The next chapter on headache, while comprehensive, concentrates excessively on organic factors and fails to offer an integrated model for the assessment and management of psychogenic headache. A chapter on psychosomatic illness and the geriatric patient provides a useful review of the difficulties encountered in interpreting physical symptoms in the elderly, although it is puzzling that the term 'psychosomatic' is defined only at the end of the chapter.

It is difficult to understand the overall purpose of this volume. Despite some useful reviews, its shortcomings far outweigh its strengths. I cannot recommend it either for institutional or personal purchase.

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Psychological Problems in General Practice. By A. C. MARKUS, C. MURRAY PARKES, P. TOMSON and M. JOHNSTON. Oxford: Oxford University Press. 1989. 406 pp. £17.50.

Psychological problems are very common in general practice but general medical training does not prepare us adequately for managing patients with them. This probably leads to much unnecessary prescription and hospital referral.

An obvious point to make about this book is that its multidisciplinary authorship provides an appropriate perspective on the nature and treatment of psychological problems in general practice – which are so closely linked to social problems and physical morbidity.

Another critical issue the book takes up is that precise diagnosis of psychological problems may not be crucial in a general practice context because this can cloud judgement on the developing problem and distract attention from the multifaceted difficulties that tend to present. Hence the book takes a problem-orientated approach and most chapters are structured in this way.

A chapter on family life-cycle emphasises the distinctive development and treatment of problems at different stages of life. A section on doctor–patient relationships gives good advice on forming a therapeutic relationship with patients. Advice on dealing with people with psychosomatic problems stresses how we need to be aware of physical disorder in these patients.

The primary-care team was mentioned, but the book seems to concentrate on the general practitioner (GP) working in isolation rather than on how problems could be managed by a team. From this point of view, some of the material has a rather hypothetical flavour. On the other hand, there is useful detail on practical procedures such as behavioural therapy, although the application of such techniques in a GPs surgery is not always possible.

I enjoyed reading this book and found it helpful It represents a valuable resource for anyone considering a career in general practice

ELIZABETH PARRY, *Academic Subdepartment of Psychological Medicine, North Wales Hospital, Denbigh*

Family Problems. By PETER R. WILLIAMS. Oxford. Oxford University Press. 1989 180 pp. £12.50.

This book is a useful short introduction to family therapy written by a general practitioner (GP) for GPs. GPs are the recipients of 'family problems' of one sort or another all the time. The author has attempted to show how a GP can usefully extend his work beyond a symptomatic response to the presenting individual into interviews with couples or whole families

The book would not be of sufficient interest to more specialised readers. For trainee GPs and more established GPs who are not used to thinking in this way, I would warmly recommend it. Useful techniques are described such as recording family genograms, and Williams' own 'family wheel', a simple method for graphically representing a family system in brief form

The book is the work of an enthusiast, not the result of research. A thoughtful evaluation of these ideas applied in practice is overdue. The idea seems so simple· 'family doctors' ergo 'family therapy'; 'index' patients leading us into the treatment of family problems Williams notes that despite our title, we GPs often know surprisingly little about families. Why is this? Williams only once mentions 'time'. He assumes a willingness to give six 40 minute sessions to the families he is treating in this way. What happens to the various relationships the doctor has with individual patients in the family after such an involvement? What of boundary issues during the 'therapy'? These and whole hosts of other interesting questions need to be examined. Only then will we begin to learn something more useful about how the techniques and approach of family therapists can genuinely enrich the work of general practice. They will otherwise remain a bit of a foreign body, not well integrated, somewhat analogous to the position of psychotherapy and general practice in the 1950s. The author is to be congratulated on making a start, along with his main GP predecessors along this route, Thomson and Hatfield.

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Treatment of Tricyclic-Resistant Depression. Edited by Irl L. Extein. Washington: American Psychiatric Press. 1990. 167 pp. £17.50.

This book originated from a symposium of the American Psychiatric Association, and some of the chapters lack conciseness, as if prepared from transcripts. There is no index, and notably missing from the contents are an account of L-tryptophan and any mention of psychosurgery. There are three chapters of obvious relevance, on monitoring of dosage and blood levels of tricyclics, on lithium as an adjunct to tricyclics, and on electroconvulsive therapy. The chapter on thyroid hormones as an adjunct to tricyclics is interesting and serves to highlight the uncertainties surrounding this treatment. How is it, for instance, that lithium, which suppresses thyroid function and thyroid hormones which replace or increase it, can augment the antidepressant effect of tricyclics? Is the effect of triiodothyronine in depression different from that of thyroxine, and are the patients who benefit only those with some degree of hypothyroidism?

Other chapters on newer antidepressants and on phototherapy seem less relevant. It was interesting to note that patients with seasonal affective disorder and summer depression have a high incidence of thyroid disease and that cold treatment (is this the same as the traditional cold morning shower?) was used to treat one such person.

I would recommend the book to trainees who have read a recent British review of the subject and want more detailed references, and to those interested in research into affective disorders.

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The Limits of Biological Treatments for Psychological Distress: Comparisons with Psychotherapy and Placebo. Edited by Seymour Fisher and Roger P. Greenberg. New Jersey: Lawrence Erlbaum Associates. 1989. 367 pp. £41.50.

Books on the shortcomings of physical or biological treatments in psychiatry are rare, but here perhaps is the acme of sustained critical rebuttal of claims which have been accepted world-wide for many years. The substance is the extensive review of published reports, and there are chapters on trials of antipsychotic, antidepressant and anxiolytic drugs, electroconvulsive therapy (ECT), and comparisons between psychotherapy and psychopharmacological treatment of schizophrenia. Drug treatment, statistical procedures, side-effects and the influence of personality on drug response are considered individually. The evidence reviewed is from earlier studies when the use of biological treatments was less sophisticated, but older methods such as exitatory abreaction and narcosis are not considered and psychosurgery is only lightly touched. More recent developments such as light therapy, sleep deprivation, lithium or carbamazepine are not included, but a special chapter is devoted to the attention deficit disorder in which the very existence of an entity is questioned.

The method of ECT is accepted as beneficial, but particular emphasis is placed on a low level of effectiveness of drugs, the limitations of the double-blind trial and the hazards of using a non-active placebo. As little is said in mitigation, at times the argument appears one-sided. Scales of measurement do not escape, and having been persuaded that they should be re-evaluated, it is not surprising to come across the view (presumably only for the purposes of research) that "the deep faith in the clinical interview as representing the ultimate criterion of status or improvement needs to be relinquished."

Condemnation of the method used does not necessarily disprove the claim itself, and the particular contribution of this substantial work is in showing the inadequacy of efforts within our present conventional framework to convince others of beliefs in effective treatment. A good case is made, but as the vast disparity between the requirements for drug trials and for psychotherapy trials are not exposed, the conclusion of the final chapter that psychosocial approaches are equal to or better than medication for the conditions for which drugs are used, seems as worthy of the 'not proven' verdict as a number of the drug trial examples.

Much of this book is compulsive reading, and whatever one's views at the end what is said here needed to be said. Its effects should repercuss through research into the effects of drugs and psychotherapy for some time.

John Pollitt, *Consultant Psychiatrist, Hayes Grove Priory Hospital*

Clinical Neurology for Psychiatrists (3rd edn). By David Myland Kaufman. Philadelphia: W.B. Saunders. 1990. 526 pp. £50.00.

This is the third edition of an American textbook which aims to serve as preparation for the US board examinations in psychiatry and neurology. If it were to have an equivalent market in this country then its readers would be preparing for the MRCPsych. examinations. So what does the book offer this group?

As a neurology text the organisation is not original. The first section contains chapters on clinical examination, basic neuroanatomy that is clinically orientated, and the principles of disease in muscle, nerve and brain. The neurological detail is appropriate for MRCPsych., but the potentially useful exercise of adding issues of special relevance to psychiatrists has limited value because of simplification in the discussion of these issues. This is particularly true of the chapter on "Psychogenic neurologic deficits", although the author makes some reparation with a brief description of neurological conditions that neurologists tend to dismiss as purely psychiatric. The second part of the book consists of 13 chapters discussing the more common neurological conditions and one chapter on neuroimaging. The clinical neurology is mostly adequately explained. The chapters on aphasias and brain tumours are written with more clarity than is sometimes the case in neurology texts, but the chapter on seizures is less clear. As in the first section of the book, the more psychiatric contributions are not as helpful as the neurology. For instance, the account of the effects of limbic system pathology is introduced by stating that; "from a neurologic viewpoint, the limbic system is the source of the libido".

At the end of each chapter there are several pages of questions and answers of multiple choice and clinical vignette type. The questions are straightforward and an aid to learning.

But is the book of use to MRCPsych. candidates or others eager to explore the field? If it were half the price some might use it as an introductory text, but the neurology can be obtained from many sources, as can similar questions and answers. The psychiatric aspects help to direct attention but are limited and occasionally misleading. The book costs £50 and I am sure that more information can be had for less elsewhere.

HOWARD RING, *Lecturer in Neuropsychiatry, Institute of Neurology, London*

Rickie. By FREDERICK FLACH. New York: Fawcett Columbine. 275 pp. $18.95.

The biography of a young person with a chronic mental illness may be enlightening, dramatic, thought-provoking, compassionate or sentimental, depending on the skill of the author. When the author is a senior psychiatrist, and the subject of the biography is the authors' own teenage daughter, one would expect such a biography to be of considerable interest to any reader, whether professional or lay. Unfortunately *Rickie* is a disappointment even though it has all the ingredients that could have enriched the literature by providing a detailed case history of the trials and tribulations of the patient and her family over a period of some 15 years.

Briefly, the biography gives an account of a 14-year-old girl who develops 'schizophrenia' and, despite years of conventional drug therapy, psychotherapy, group therapy and prolonged periods in hospital, only showed sustained improvement following treatment for an eye defect by a developmental optometrist followed by megavitamin treatment for a low blood histamine concentration! The traumatic effect of caring for a mentally ill teenager on the family was largely responsible for the parents getting divorced and the consequent adverse effects on the three normal children. I had hoped that this book could be recommended to a lay person who had to grapple with the emotional problems of caring for a mentally ill relative, a book that would give them a realistic account of how mental disease is treated and why, in general, the prognosis is good. Instead, the lay reader is given a dramatic account of the litany of confused and contradictory treatments and diagnoses to which the patient is subjected. Finally, as conventional psychiatric treatment fails, 'alternative' medicine steps in to resolve the problem.

There is a very real need for a book written for the lay person by a psychiatrist that would help the reader to understand the clinical problems of diagnosis and treatment of serious mental illness like depression and schizophrenia. This book fails to fulfill such a role and may even lend credibility to those advocating an irrational approach to the treatment of the mentally ill.

B. E. LEONARD, *Professor of Pharmacology, University College Galway, Ireland*

Something Sacred: Conversations, Writings, Paintings. By MARY BARNES with ANN SCOTT. London. Free Association Books. 1989. 146 pp. £9.95 (pb), £25.00 (hb).

Mary Barnes, as most readers will know, was the subject of a previous book – *Mary Barnes: Two Accounts of a Journey Through Madness* – in which she provided one account and her therapist, Dr Joseph Berke, the other. Once again she has travelled into print with a companion, in this case Ann Scott who at the time was an editor with the present publishers whose motto is "an association in which the free development of each is the condition of the free development of all".

The first 80 odd pages of the book are derived from taped interviews with Ms Scott, and the subsequent pages contain poems and reproductions of paintings. Ms Barnes' art is vigorous and colourful, but is not carefully shaped. Ms Scott attempts to provide a shape to the biography, but her carefully thought out questions more often provoke agreement from Ms Barnes than further information. Early in the book Ms Barnes describes one of her last meetings with her mother: "It was a realisation to me that she was in her world and it was not for me to try to bring her into mine, but to respect hers and leave it at that. I had learnt. . . to accept a gap".

It becomes clear that Ms Barnes' talent, put to good effect in the well known regression which is reported in the earlier book, is her ability to accept what happens to her with a minimum of intellectual resistance. As one reads on, one comes to respect her for this, and to respect the good faith of the therapists who fostered it without exploitation. Acceptance does not make for acute observation, however, and even the description of Kingsley Hall, the fulcrum around which her life turned from nurse-tutor to celebrated former mad-woman, lacks the sort of detail that many curious psychiatric readers will probably have wanted.

I cannot recommend this book highly to the general psychiatric reader. Not that this will influence its sales: Mary Barnes' celebrity as the icon of the antipsychiatry movement will remain secure.

DIGBY TANTAM, *Professor of Psychotherapy, University of Warwick*

Legacy of Silence: Encounters with Children of the Third Reich. By DAN BAR-ON. Harvard: Harvard University Press. 1989. 338 pp. £19.95.

"Children whose parents witnessed or took part in the persecution or extermination of Jews and/or Gypsies and who are willing to participate in a research project of an Israeli psychologist at the local university please. . . ."

Would you have answered it? We all have relationships with our fathers. Some of the subjects of this book did not know their fathers; some knew them well. Some felt loved by them; some thought they were going to be killed by them. Some of the fathers committed suicide and some were executed. All were Nazis.

This book includes statements from interviewees who had spent years trying to push this bit of family background out of their minds; and some who talked at length and clearly felt a need to go on doing so. It reports statements about Nazi parents who said they could not go on, and who did or did not. Bar-On reports that Himmler himself had found it hard to stomach watching a mass firing squad so had invented gas chambers to make the task of extermination more comfortable. Himmler's daughter refused a meeting and was later reported to be involved in neo-Nazi activities.

One of the interviewees married a Jew, one became a priest and heard confessions from Nazis. One became a cabaret singer and repeats some of the anti-fascist songs of the 1920s and 1930s.

When the research was finished, Bar-On organised a conference at the University of Wuppertal and invited the 50 interviewees. Some of them came, but, to maintain anonymity, name-tags were dispensed with. A Dutch delegate, the child of a collaborator, introduced himself and suggested that the others do likewise. They did. The interviewees thus met each other for the first time and later went on to ask the Dutch members to help them set up a new self-help group.

This was not an easy book to read or to review (I found the shorter *Born Guilty* by Peter Sichrovsky (Tauris, 1989) to be easier), but it is as compelling as many of the works I have read related to the holocaust. The Jew cries with the German and has at least one reader in tears, but of course it does not answer all my questions. Indeed, it has raised a new one. I started this review with a question to you, the reader. Here is another one. One interviewee contemplates how much worse the holocaust would have been if it had been perpetrated by his generation: "We were trained to hate from a very early age". He remembered having said: "Mama, I would like to be in the Hitler Youth too." The question: how diligent a member of that youth club might you or I have been?

PETER BRUGGEN, *Consultant Psychiatrist, Hill End Hospital, St Albans, Herts*

The Plural Psyche: Personality, Morality and the Father. By ANDREW SAMUELS. London: Routledge. 1989. 253 pp. £12.95 (pb), £25.95 (hb).

I have presumably been asked to review this book in my capacity as a Jungian; this book has already had one review in these pages. This book makes a great deal of sense to me and it seems relevant to important depth psychological controversies and even to general political arguments of the present day, such as the debate about forms of government – authoritarian, bipartisan, or pluralist democratic. For a start, the author's contention that politics and political factions reflect important part 'selves' of the human personality seems self-evident to me, but may be new or nonsensical to some readers.

Samuels asks how to link the monotheistic/integrative/unus mundus/elitist concerns of the classic Jung with the polytheistic/interactive/microscopic/democratic concerns of the post-Jungians. For Samuels, pluralism might meet this challenge, which is not of course a narrowly Jungian conflict, but could be and to a certain extent is formulated in much broader human social and political terms. He feels passionately that pluralism seeks to hold a dynamic balance between unity and diversity, and that diversity need not be a basis for schism. However, he does not successfully examine the question "when does pluralism become schism, either between the different 'selves' of the human individual or in the case of human groups?" To a certain extent, however, he does answer the question "having many internal voices, how does one speak with one voice?" through his own personality, which binds the book together in a refreshing whole. Personality, he says, at any one moment, is the outcome of competition between the various parts of the psyche. But the examples of the interaction of parts of the psyche which he gives are practically all opposites (dualities and polarisations). Dualisms, even polarisations, are to be celebrated, profited by and learned from. To caricaturise his approach, he looks at areas of schism or conflict in the group or the individual, and singles out and highlights an important pair of opposites. Samuels' way of looking at conflicts or schisms, often by turning them inside out or on their heads and offering new ways of resolving or using them, is original and stimulating. The polarisations he deals with in this way include nature versus nurture, or archetypal versus developmental schools in analytical psychology, with his own concept of the 'imaginal network'. Clock time versus psychic time is a subheading of this pair of opposites. Another major theme is that of the parts of ourselves which have to do with sex, gender, and relating. The Freudian primal scene and the Jungian union of opposites, the father and the masculine in us, the mother and the feminine in us, the modern father's role, and the use of primal scene fantasy as monitoring or indicating the progress of therapy are all treated in a way which makes a lot of clinical sense, with plenty of food for thought.

Although apparently suspicious of using case-history vignettes (we all know they prove nothing), when he does use them it is in a way which succinctly illustrates what he is talking about. Fortunately, he is not similarly suspicious of the selective use of numerous quotations, although the surfeit of these might be too much for some, and give the impression that he is trying too self-consciously to be scholarly.

Another pair of opposites which he discerns is that between the black-and-white absolutist morality and the moral imagination that we all need to live with ourselves and particularly with each other. He calls the black-and-white morality "original morality", thereby linking it with the related concept of "original sin", and although the reader is all the time reminded of the Kleinian paranoid–schizoid and depressive positions, the author is emphasising that what he is writing about is not that. I should say the reader has as much right as the author to decide on that point. In fact, this chapter is all the more valuable and interesting for that very link.

The main symbol used by Samuels for that part of ourselves which is concerned with using these oppositions, schisms and competing motivations for our own creative or selfish purposes, as well as for the good of mankind, is the god Hermes, perhaps the opposite of being stuck, fused or parthenogenetic, and the antithesis of the parents being two halves stuck together, rather than two individuals relating in love or whatever. The union of the parents and the *coniunctio* of opposites is celebrated, not envied and rubbished by Hermes. Hermes is the part of us that transcends or transforms (I cannot find the correct word) primal-scene envy to primal-scene liveliness and interest and personal creativity. Being a clever entrepreneur, he always manages to make a good thing out of it, first and foremost for himself.

Incidentally, the book made me feel like an ignorant clodhopper who wanted to tell the author he was too clever by half. I was gradually able to take a bit of comfort in coming to the conclusion that the author is identified to a certain extent with the hero of his book, Hermes. Having understood this, I was much more free to enjoy the freshness, the genuine helpfulness and the intellectual vigour of this book I consider it extremely good value for money and for time spent in understanding it.

J. REDFEARN, *Jungian Psychotherapist, 38 Frognal, London*

Psychoanalysis and Psychosis. Edited by ANNE-LOUISE S. SILVER. Madison. International Universities Press. 1989. 584 pp. $60.00.

This is a book which most psychiatrists will find well worth reading. The title is in a way misleading because it is as much concerned with clinical problems which psychiatrists of all theoretical persuasions face as it is with psychoanalysis. The book grew out of the 31st Annual Chestnut Lodge symposium which also commemorated the 50th anniversary of the arrival of Frieda Fromm-Reichmann at the Lodge. There are 26 contributions from distinguished psychiatrists who trained and worked there between 1935 and 1957, the year of Fromm-Reichmann's death.

In the years immediately following 1945, Chestnut Lodge became internationally known as a centre for the psychoanalytic and psychotherapeutic treatment of the functional psychoses – for those cases of schizophrenia in particular which had failed to benefit from insulin coma treatment and electroconvulsive therapy This reputation was almost entirely due to Fromm-Reichmann, a German psychoanalytically trained psychiatrist who had emigrated to the USA and joined the Lodge in 1935. With her arrival, a series of publications began to stream from the Lodge and this continued for more than 25 years. Many of these were written before the introduction of the psychotropic drugs. They confirm Bleuler's observation that the symptoms and course of schizophrenic psychoses are profoundly influenced by external events and the psychical reactions to which they give rise.

The book is essentially a dedication to the memory of Fromm-Reichmann as a psychiatrist, a psychoanalyst, a teacher and friend. Will writes a chapter "In Memory of Frieda", Schultz on "Recollections of supervision with Frieda Fromm-Reichmann", Silver & Curry on "Fromm-Reichmann's contributions at staff conferences", and Dyrud on "The early Frieda and traces of her in her later writings". Special mention must be made of the contribution by Weininger who joined the staff of the Lodge in 1936. For those who worked in mental hospitals before 1952 this chapter will evoke recollections of the serious problems of management which daily confronted the medical and nursing staff.

Those who contribute to the section entitled "Psychoanalytically orientated treatment of schizophrenia" agree that the gradual introduction of psychotropic drugs altered the atmosphere at the Lodge as it did in all psychiatric hospitals. As Gibson states, the use of these medications does not have to lead to a mechanical approach to the treatment of the schizophrenias. Gibson and the others who write in this section have found that psychotherapeutic work which pays attention to transference manifestations can proceed satisfactorily if medication is kept to a minimum. The value of this approach lies in the fact that in time this medication can be discontinued and the natural trend towards recovery which is present in at least 50–60% of cases given the diagnosis of schizophrenia, can continue under its own momentum.

This review would hardly do justice to the contents of the volume if mention was not made to the sections on "Psychoanalysis of non-hospitalised patients" and that on "Applications in history and literature". The book, by former pupils of Fromm-Reichmann, is a true recognition of her special contribution to an important branch of clinical psychiatry.

THOMAS FREEMAN, *formerly Consultant Psychiatrist, Holywell Hospital, Antrim and the Anna Freud Centre, London*

Archetype: A Natural History of the Self. By ANTHONY STEVENS. London: Routledge. 1990. 324 pp. £12.99.

Stevens is both an experienced Jungian analyst and psychiatrist and this exceedingly rich book of his, originally published in 1982, has been reprinted in 1990. The interest is enhanced by the fact that he cross-fertilises the ideas of many specialist disciplines including psychiatry, psychology, ethology and biology. His main theme juxtaposes Jung's theory of archetypes with the work of the ethologists and theories of brain activity. Stevens acknowledges the detailed analytic work and behavioural studies of the ethologists but sees these as having some limitations. He thus links Jungian ideas to ethology to expand the concept of inner processes with symbolic content.

In the first section of the book he draws parallels between the innate releasing mechanisms in animals and man, which ensure adaptive responses for survival, and processes in the mind. These processes are the archetypes, the innate psychic predispositions of human beings to relate actively to others to survive and develop a sense of identity and well being. This discussion is made more fascinating when it is realised that Jung described the archetypes as biological entities (p.61). Stevens clearly describes how, for example, a new born baby and its parents both have these innate archetypal patterns which help them to respond to each other. In developing the links to biological functioning he makes the interesting suggestion that the theory of the two hemispheres of the brain, each displaying different modes of functioning, corresponds to Jung's idea of the self-regulating nature of the psyche and the continuous process through life of achieving the *coniunctio* of the opposites. An extension of this idea is the discussion that both sexes are born with primitive masculine and feminine characteristics which are differentiated through life's experiences, and that the *coniunctio* may ultimately provide the individual with a creative identity.

Stevens writes in an enquiring way but also hopes to influence the reader. He finds many of the pieces of research which he quotes fascinating and so avoids presenting a mystical view of Jung, which is a frequent misunderstanding of Jungian views. The book is very worthwhile reading as Stevens produces a clear elucidation of archetypal theory and also demonstrates its sound clinical and practical application.

ELIZABETH GEE, *Professional Member, Society of Analytical Psychology, London*

Trancework: An Introduction to the Practice of Clinical Hypnosis (2nd edn). By MICHAEL D. YAPKO. New York: Brunner/Mazel. 1990. 480 pp. $42.50.

The "rich and complex world of clinical hypnosis" could have no more worthy an introduction than the second edition of this book which is divided into two sections, a "Conceptual framework" (11 chapters) and a "Practical framework" (12 chapters). There are 20 tables, a useful glossary, bibliography and a name and a general index.

New features include chapter 19, "Hypnotic trance-scripts", in which six accounts of sessions with clients are described. These cover self-definition, pain management, goal orientation, adaptability, weight management and stress management and are designed to guide the reader in general treatment. The cases serve ideally as models for the many similar problems which commonly present, and the author emphasises the need to tailor the treatment to the individual.

Chapter 20 indicates the usefulness of hypnosis for coping with terminal cancer. Again, presented as a trance-script, integrating the induction experience with therapy, it demonstrates the value of kindness and empathy in alleviating emotional trauma and physical discomfort.

Some chapters include interviews with experts in the field (under the subheading "frames of reference"), which add interest to the discussions of the conduct of cases. Special suggestions of items "for discussion" and of "things to do" are original and helpful. Most chapters end with a summary or with conclusions – again, useful additions to any teaching publication.

The author possesses an unpretentious and flowing style which allows for easy reading by any psychotherapist wishing to gain an understanding of some of the techniques and processes of hypnosis. Although certain chapters may be oversimplified, each is succinct and thought-provoking. It is curious to note the title and the frequency of use of the word 'trance' – a term perhaps even less definable than 'hypnosis'. Nevertheless, there is no doubt, as stated in the introduction, that every specialist volume such as this should "represent an effort to make the acquisition of skills to apply hypnosis more easily accomplished by the reader." Has the author succeeded in this ambition? I think so. But in the acquisition of such skills, understanding and experience are essential.

Trancework makes good reading for the beginner as well as for the expert. I recommend it.

DAVID WAXMAN, *retired Associate Specialist in Psychiatry, Central Middlesex Hospital, London*

Storymaking in Education and Therapy. By ALIDA GERSIE and NANCY KING. London: Jessica Kingsley Publishers. 1990. 407 pp. £29.95.

The authors have drawn myths and tales from all over the world and grouped them around seven themes, all reflective of forms of human experience. They suggest ways in which these may be used to develop the imagination's original and unique voice through therapeutic encounter in group work. The book is written for teachers, therapists, pastoral counsellors and group workers.

This volume presents both the material and the method of extending the use of creative imagination to bring it to bear more fully on some of the major problems which we all face. The stories themselves are divorced from reality, many being played out by a variety of animals, and thus enable the participants to 'play with' ideas and emotions which might be too threatening to be faced more directly. It enables them to escape from the tight categories of rigid thought patterns into which their own cultural upbringing may have imprisoned them.

Life itself tends to be lived in short-hand. We communicate in short-hand and eventually tend to think and live in short-hand. This saves a great deal of time and may work well for the ordinary situation in which we find ourselves. However, it does fall down when we meet an entirely new situation. At that time we need to find some way of escaping from the categories, extending our inner emotional responses, and examining the situation in long-hand. We may be helped to do this by moving into an entirely new environment in which none of the normal categories function. The imagination is captured and we begin to extend our categories and become more able to cope with the new experience.

A textbook for group work.

LOUIS MARTEAU, *Consultant Psychotherapist and Director, The Dympna Centre, London*

Reaching the Affect: Style in the Psychodynamic Therapies. By EMANUEL F. HAMMER. New Jersey: Jason Aronson. 1990. 232 pp. $25.00.

This book focuses on an aspect of psychotherapy – the actual language used in communicating with the patient. It provides a guide to an area not often addressed: how do we speak to patients and how do we get the meaning across so that the patient feels the importance of it? Can we translate into an easily digestible but concentrated form our thoughts, feelings and those that have been communicated to us. Among all the activities that go on in psychotherapy, at some stage there will be a need to return to the patient his various communications powerfully enough to connect with and reach him or her on an affective level. Hammer's book is about how this can be achieved. He discusses the similarities that writers have with psychotherapists, both being interested in exploring human relationships internally and externally and finding words to express them. He suggests that the most concise form is that of poetry, full of imagery and metaphors, and that we can learn a lot in the way of style from literature; "from the creative writer, therapists can learn communicative style – a finely chiselled clarity ... and the impact as in good poetry". The book explores the fundamentals of style and then the differing types: poetic, earthy, creative and playful. This for example involves discussion of the use of humour and jokes in therapy.

I started the book with the prejudice that it seemed limited to consider psychotherapy concentrating on what the therapist should say, as if to be successful in treating patients one has to rely on getting the words just right. What about the other means of communicating through the transference or projective identification? However, as I read on it was not the case that these were ignored but that Hammer was concentrating on 'how to say' when it is necessary. The book contains clinical material and in it Hammer exposes some of his own style. The book was enjoyable and easily read and to be recommended to all those involved in psychotherapy

ANDREW SOUTTER, *Senior Registrar in Psychotherapy, Claybury Hospital, Essex*

Resolving Resistances in Psychotherapy. By HERBERT S. STREAN. New York: Brunner/Mazel. 1990. 312 pp. $22.95.

Strean, Director of the New York Center for Psychoanalytic Training, has produced a clinically useful book on the resistances in psychotherapeutic work. The identification, analysis and resolution of resistance as well as the understanding of its purpose has occupied psychoanalysts and psychotherapists from the time of Freud's seminal works through subsequent psychoanalytical developments to the present day.

Whatever theoretical orientation in the technique used in any psychotherapy, the observation that patients, despite the suffering that brings them to treatment, resist efforts to ameliorate this suffering is universal. It is indeed only psychoanalysis that has addressed this problem. In certain forms of therapy, if the patient fails to improve or indeed deteriorates, the technique in use is questioned and an alternative therapy offered rather than exploring the significance and meaning of the tenacity of symptoms. While it is relevant to question whether one has applied an appropriate form of treatment when negative therapeutic reactions arise, it is equally relevant to explore what possible purpose the negative therapeutic reaction is serving for the maintenance of the patient's mental equilibrium. More importantly, one needs to address the problem of whether the resistance is essential to the patient's psychic survival.

Strean defines resistance and its defensive functions in terms of psychic danger with respect to the types of resistance via repression and defence mechanisms. He also describes transference resistance. He offers a review of the literature from the Freudian, neo-Freudian, and non-Freudian schools. He omits the Kleinian school and its body of work on the negative therapeutic reaction which would have added a further dimension to the review, in particular the work of Herbert Rosenfeld.

Strean explores resistance and its development from the beginning of treatment through to the resistances of termination. He uses a wealth of clinical material that elucidates many of the pitfalls arising in treatment when resistances are not recognised or analysed.

Written with a refreshing lack of jargon, this book will provide an extremely useful guide for students. Those already experienced in these disciplines may find it a little lacking in depth.

BERENICE KRIKLER, *Principal Clinical Psychologist, Portman Clinic, London*

Artaud on Theatre. Edited by CLAUDE SCHUMACHER. London: Methuen. 1990. 210 pp. £25.00.

The life and work of Antonin Artaud casts an interesting light on the relationship between madness and creativity. Artaud, founder of the 'Theatre of Cruelty', was one of the most important theorists in 20th-century drama. His ideas, which centre on the importance of treating theatre as a plastic medium in which the text should be no more important an element than any other, have had a crucial influence on the contemporary approach to staging and performance. This selection of his writings is somewhat misnamed; as well as theatre, his views on cinema, religion and sexuality, among other topics, are well covered.

In his commentary Schumacher, while admitting Artaud's lifelong addiction to laudanum and the personality problems which caused him to fall out with practically all his patrons and collaborators, coyly tries to suggest that his mental illness may be in doubt. However, the evidence to the contrary from both the writings and the biographical notes seems quite clear. Between 1941 and 1943, for example, he called himself 'Nalpas' and believed 'Artaud' to have died of malnutrition and poisoning in 1939.

Even during his long hospital stay at Rodez, however, when he was unable to write about theatre, his letters retain the extraordinary power and lucidity that characterise his essays on dramatic theory. It is arguably the force of his writing, and the extremes to which he pushed his ideas, which have made his work so influential. The ideas themselves are reasonably straightforward; it is the outrageous way in which they are expressed that grabs the attention.

This book is thoroughly recommended on two counts: as an introduction to some of the most exciting writing ever published on theatre, and as a fascinating insight into the disordered mind of a man of great intelligence and creativity.

DAVE JAGO, *Publications Manager, British Journal of Psychiatry*

The Nature of Adolescence (2nd edn). By J. C. COLEMAN and L. B. HENDRY. London: Routledge. 1990. 255 pp. £9.99.

This is the updated second edition of an aptly titled and popular book written by authors well qualified in the field. John Coleman, a psychologist, is the Director of the Sussex Youth Trust, and Leo Hendry is the Professor of Education in Aberdeen.

Although brief, this text covers the biology, psychology and sociology of adolescence broadly and from a number of complementary perspectives. It is aimed at professionals who work with young people from a variety of disciplines. This book has a clear purpose and scheme running through it. In structure its contents could be likened to an intellectual sandwich – of theory at beginning and end separated by a generous filling of empirical research evidence.

The nine chapters begin with an introduction which summarises three theoretical approaches – psychoanalytic, sociological and lifespan developmental psychological. Empirical evidence is produced in abundance in the seven chapters that follow on the physical, cognitive, self-concept and sexual arenas of adolescent development. There are chapters on the family, friendship and peers, and a new one on work, unemployment and leisure, which considers the transition from school to adult society.

In the final chapter the evidence cited in preceding ones is used to evaluate the initial theories. Each is seen as having value but as open to criticism. While the serious problems which a significant proportion of young people experience in growing up are not diminished (over a quarter are cited as reporting higher levels of psychological stress), the authors conclude that the majority of young people handle the major transitions of adolescence in a relatively smooth way. They agree with the conclusions from a series of studies in the last 20 years which do not support the notion of normal adolescence being a period of storm and stress. The 'traditional' psychoanalytic and sociological and more recent lifespan theories are seen as providing the foundation for understanding the normal development and resilience of the majority. The authors conclude by evaluating Coleman's 'focal' theory which suggests that resilient adolescents are those who are able to face the challenges posed by the developmental transitions in growing up, and deal with them one at a time and in social circumstances which do not excessively compromise their feelings of self-control of events. The criticisms of this approach are argued.

This slim volume is packed with information. Most of the chapters read well although I did find the material condensed and demanding of concentration in several sections. The thirty pages of references are a major asset in themselves and the whole is excellent value for money. I would regard this book as essential reading for all professionals who work with young people.

RICHARD WILLIAMS, *Consultant Child and Adolescent Psychiatrist, Bristol Royal Hospital for Sick Children*

Growing Old: The Ultimate Freedom. By MAXWELL JONES. New York: Insight Books. 1988. 116pp. £15.90.

One of the many privileges of working in old age psychiatry is the opportunity it affords to gain a longitudinal view of life. Now over 80, Jones, well known for his pioneering work in adult psychiatry, uses his life experience to give an uninhibited account of growing old. He draws on his work in therapeutic communities and its extension to other social systems to question the validity of many of our Western cultural stereotypes regarding the process of growing old.

He chooses five topics of particular relevance to the elderly and, with a mixture of anecdote and home-grown experience, leads us willingly on a quest to find a 'deeper reality' and 'the meaning of life'.

Retirement he re-names 'freedom'. Life up to this event is largely a matter of conformity. This freedom enables the 'retiree' to escape the restraining influences of society and allows time to seek a deeper reality. However, to achieve this, hobbies are insufficient. A peer group which facilitates interaction of like-minded individuals is a must, irrespective of age.

Education in its orthodox form is a one-way process aimed primarily towards our active participation in a materialistic society. It offers little in the way of social learning or personality growth and inadequately prepares us for living or dying. He highlights the value of a Socratic approach as well as the role of supportive groups from childhood to old age. The often used perjorative phrase 'second childhood', he positively re-frames as a second chance to grow and develop, this time without interference.

Western culture, which predominantly favours youth, conspires to isolate the elderly. The resulting loss of social intercourse is a minor form of sensory deprivation which we know can lead to abnormal mental states.

In a discussion on "Communities as support systems" he raises all the obvious difficulties which are part of growing old: physical disability, loss of status and role, loneliness and isolation. These losses, and in particular the social isolation experienced by so many elderly, can be counterbalanced by groups (communities) that facilitate intimacy and sharing.

In a moving chapter on "Death dying and the aftermath" he quotes the work of Cicely Saunders and Kubler Ross to weave a rich tapestry of exploration filled with optimism, growth and joy.

Before reading his thoughts on "Spirituality" we see him sitting in his Nova Scotia home, inspired by the view of nature outside his picture window. Scientific reductivism and rational thinking have lead us up a cul-de-sac where spirituality is repudiated and death is the logical end. Jones cites two incidents where he felt part of an awareness beyond self, the five senses, time and space and experienced an unexplicable feeling of exaltation and joy.

In this chapter many of the ideas already explored come to fruition. Creativity, spirituality, intuition and self-reflection, those latent potentialities glimpsed at in childhood, can be rediscovered in old age.

This is a book filled with peace, contentment and a joy beyond the objective reality. Not only is it a valuable contribution to our understanding of growing old but it will, I hope, help many of us prepare for the ultimate freedom.

CON KELLY, *Senior Registrar in Old Age Psychiatry, St Charles' Hospital, London*

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